American Journal of Diseases of Children

VOLUME 32 OCTOBER, 1926 NUMBER 4

THE VALUE OF ELEMENTARY PHOSPHORUS IN RICKETS

AN EXPERIMENTAL STUDY *

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REVIEW OF THE LITERATURE

The idea of employing elementary phosphorus in the treatment of rickets had its origin in the investigations of Wegner [1] about 50 years ago. At that time, acute phosphorus poisoning was not infrequently seen by the pathologist, due to its use for suicidal purposes. As Wegner quaintly puts it: "A gulp of the morning coffee in which fluorescent, demoniacal match tips have been dissolved, taken in the dark, melancholy kitchen, affords a far easier death than a leap into the cold water." In the course of necropsies on such subjects, Wegner encountered an occasional instance of chronic phosphorus poisoning associated with hardening and enlargement of the bones, instead of the usual softening or necrosis. Observations of this kind induced him to undertake an experimental study of the effect of minute quantities of elementary phosphorus on the bones. In the course of these experiments he found that the bones of rabbits and chickens, which were fed about 0.00015 Gm. of phosphorus daily, showed a "phosphorus layer," a transverse band in the subepiphyseal region. This peculiar phenomenon could be brought about only in growing animals. The layer was so distinct that Wegner stated it could almost be grasped with the hand. In the region in which normally the preparatory cartilage merges almost insensibly into wide meshed trabeculae of bone, a tissue was formed which, even when examined with the unaided eye, appeared dense and compact, resembling rather the cortex of the hollow bones. Under the microscope, this "phosphorus layer" was found to consist of well formed bone, with the marrow spaces greatly reduced, and occupying an area a few millimeters

* Received for publication, June 1, 1926.

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1. Wegner, G.: Ueber das normale und pathologische Wachstum der Roehrenknochen, Arch. f. path. Anat. **61**:44, 1874.

in depth, adjacent to the zone of growing cartilage. He believed that the development of this bone from cartilage had proceeded in the normal way, the peculiarity being that the proliferated cartilage cells had been changed into bone, rather than into lymphoid marrow. As a result of this abnormal ossification, the "spongioid bony substance" at the end of the diaphysis was replaced more or less by a compact bony mass. Although the periosteum seemed normal to the naked eye, the microscope disclosed that bony masses had been laid down at this site.

Wegner was exceedingly cautious in drawing an inference between these experiments in which ossification had been brought about by feeding phosphorus, and a therapeutic application of elementary phosphorus in the faulty ossification of infantile rickets. He writes: "The solution of the question of the influence of phosphorus on rickets must be left to the future," and again, "the matter is different in relation to rickets. Theoretically viewed, it does not seem probable that one has found a cure for this disorder in phosphorus." Indeed he states that, if one gives phosphorus and at the same time decreases the intake of inorganic substances, especially of calcium, the bones undergo a change "which absolutely resembles that which we are accustomed to term rickets in man."

A few years later, Gies [2] repeated Wegner's experiments, using small doses of arsenic in place of phosphorus, and showed a similar apposition of bone adjacent to the epiphyseal cartilage in rabbits, chickens and young pigs.

The application of these studies to infantile rickets is due to Kassowitz [3] of Vienna, who for many years and with great zeal carried out experimental investigations and clinical studies on this subject. In 1884 he published his well-known thesis which consisted of two parts, the first detailing the results of animal experiments in which small amounts of elementary phosphorus were added to the dietary. His conclusion was a complete confirmation of Wegner's investigation. In the second part, he reported a large series of clinical observations with the use of phosphorus in the treatment of infantile rickets, and concluded that we possess in phosphorus "a specific remedy for rickets, and that elementary phosphorus given in small and harmless doses has shown itself to be a direct, rapid and reliable therapeutic measure for rickets and its individual symptoms." At the beginning he dissolved it either in almond oil or in olive oil, but as it did not keep well and "also on account of its being less expensive," he writes, "I changed in the year 1881 for the outpatient department to a solution of phosphorus

2. Gies, T.: Experiment. Untersuch. ueber den Einfluss des Arsens auf den Organismus, Arch. f. Exper. Path. u. Pharmakol. **8**:175, 1877.

3. Kassowitz, M.: Die Phosphorbehandlung der Rachitis, Ztschr. f. klin. Med. **7**:36. 1884.

and cod liver oil; cod liver oil 100, phosphorus 0.01, of which 1 to 2 teaspoonfuls are given daily." He gives his dosage as 0.00015 Gm. a day. In looking over the case reports of Kassowitz in the light of our recent knowledge of the pathogenesis of rickets, it is suggestive to note how large a number of infants in whom he brought about a cure were treated during the summer months. Hryntschak, a young assistant of Monti, remarked on this coincidence at a meeting of the Vienna Medical Society, and suggested that "the favorable season of the year" might have played a rôle in the curative process. In view of subsequent events, it is instructive to note the following dogmatic statement of Kassowitz: The changes which came about in the rachitic cranial bosses and in the large fontanel are so remarkable and rapid that there can be no question whatsoever of chance, or of an application of the "post hoc, ergo propter hoc" syllogism.

Naturally, the phosphorus treatment was immediately tried in the various clinics of Europe. Hochsinger [4] of Vienna reported in the following year that "we possess in phosphorus a specific remedy for rickets. In phosphorus, we have found a remedy for rickets which, in regard to rapidity of cure and reliability of action, greatly surpasses all the known methods of treatment of rickets." Heubner and Biedert agreed with Hochsinger in this conclusion, as did Rauchfuss of St. Petersburg. There were, however, some dissenting voices, especially one from the Charité Clinic in Berlin, a report by Schwechten [5] which concluded with the verdict that "phosphorus is no specific for rickets." There was a memorable "phosphorus debate" held in the Vienna Medical Society during four successive meetings of the year 1885, the upshot of which was that phosphorus seemed to be a specific for rickets in Vienna but of no value in Berlin. A copious literature on this subject developed between the years 1885 and 1895. There was a slight revival of interest in 1900, occasioned by the articles of Kassowitz, of Epstein and of Schabad, which added the new aspect that excessive doses—which had been given by mistake in several instances—could result in death.

In this country the phosphorus treatment of rickets was likewise adopted. Jacobi [6] advocated it warmly. In view of the present high estimation of cod liver oil, it is interesting to record his statement that "phosphorus given simultaneously with cod liver oil is a good combination, but it is a doubtful practice, so long as the latter is not a uniform compound, to dissolve phosphorus in the oil." The

4. Hochsinger, C.: Die Phosphorbehandlung der Rachitis im Jahre 1884, Jahrb. f. Kinderh. **23**:91, 1885.

5. Schwechten, E.: Zur Phosphorbehandlung der Rachitis, Berl. klin. Wchnschr. **21**:825, 1884.

6. Jacobi, A.: Therapeutics of Infancy and Childhood, Philadelphia, J. B. Lippincott Company, 1903, p. 131.

literature abounds with expressions of approval as well as of disapproval of the phosphorus treatment. If we consult the textbooks of pediatrics and of therapeutics, we find that there is no well defined opinion on this question today; most of the authors take a mid-position —not claiming a therapeutic action for phosphorus, but, nevertheless, mildly recommending its use. In some texts phosphorus is highly recommended as a cure for rickets.

In 1921, Phemister and his co-workers [7] published a roentgenologic study "on the effect of phosphorus in rickets." This investigation was based on a series of patients who had been treated with cod liver oil and phosphorus, or with phosphorus alone. Two rachitic children were given merely phosphorus and were cured; it is to be noted, however, that the treatment was carried out during the summer months. The authors conclude that "The end-results produced by phosphorus, and the combination of phosphorus and cod liver oil were quite similar. The rarefied areas of the rachitic bones acquired an approximately normal density in both instances." . . . "While they (the results) suggest that phosphorus alone may cause equally prompt healing of rickets, further observation will be necessary to clear up this point." Very recently, experiments have been carried out by Bernhardt and Kabl [8] in regard to the rickets-productive properties of phosphorus, to which, as has been stated, attention was drawn in the original study of Wegner. These investigators used as their standard dietary a ration containing 1 per cent of cod liver oil in which the phosphorus was dissolved. From these tests it was concluded that white phosphorus, added to a ration poor in calcium and phosphorus, leads to "rachitoid" lesions.

EXPERIMENTS

For the past two years we have been carrying out a series of feeding experiments to determine the influence of white phosphorus on the development of rickets in young rats. In this work, we have followed the technic which has been employed in the course of the investigations which have been reported during the past years from this laboratory. Various basic diets have been employed. In the main, the standard rickets-producing dietary was fed, that is, the Sherman-Pappenheimer diet, which is low in phosphorus and high in calcium. As a supplement, small amounts of a 1 per cent solution of phosphorus in linseed oil was given by mouth. This solution was diluted with linseed oil in the ratio of 1:5 and 1 drop was fed daily by pipet. Each animal received,

7. Phemister, D. B.; Miller, E. M., and Bonar, B. E.: The Effect of Phosphorus in Rickets, J. A. M. A. **76**:850 (March 26) 1921.

8. Bernhardt, H., and Kabl, C. K. H.: Exper. Stoerungen des Mineralstoffwechsels und ihr Einfluss auf die Wirkung des weissen Phosphors, Ztschr. f. klin. Med. **102**:147, 1925.

therefore, approximately 0.2 mg. of white phosphorus a day. At the outset an attempt was made to give phosphorus in somewhat larger amounts, diluting the preparation with only 3 parts of linseed oil. It was found, however, that when this large amount was fed, the majority of the rats died within a fortnight. It has been our experience, however, as well as that of others, that the dose can be augmented considerably if it is made small initially and increased gradually.

We were able to confirm the observations of Wegner and other investigators to the effect that phosphorus leads to the development of a band (Phosphorschicht) at the epiphyses of the ends of the long bones. This transverse band is apparent at the end of the femur or of the tibia, even on gross examination when the epiphyses are sectioned longitudinally. This "phosphorus layer" also stands out sharply in

Fig. 1.—Roentgenogram of rat which had received the standard rickets-producing diet plus elementary phosphorus. Co-existence at knee joints of rickets and of subepiphyseal "phosphorus band."

roentgenograms of the knee joint, appearing as a distinct, opaque, rather sharply circumscribed transverse band situated near the epiphyseal line (fig. 1). It was noted in one instance after phosphorus had been fed for sixteen days. Microscopically, the band shows itself to be composed of an area of trabeculae of bone adjacent to the zone of preparatory calcification, the layer of actively proliferating cartilage. These trabeculae are strong, mainly longitudinal, with small intervening marrow spaces and occupy the site of the primary, rather than that of the secondary spongiosa. Judging from the manner in which they splinter on sectioning, the ossification would seem to be abnormally dense; this calls to mind the condition which has been found in the "lumpy jaw" of chronic phosphorus poisoning. With a dietary such as 84, which is deficient in several respects and leads to osteoporosis as well as to rickets, this formation of a layer of bone within the marrow is particularly striking (fig. 2). The cortex is likewise thickened at

times, but to a less extent. The main shaft of the bone, both in cortex and marrow cavity, remains unaffected.

As stated, the development of this subepiphyseal layer had been established by the investigations of various authors. The question which interested us was rather whether phosphorus, in virtue of its bone-forming properties, is able to avert the development of rickets. In the large series of animals which was fed the standard rickets-producing diet 84, all without exception developed rickets (fig. 2) and apparently to the same extent as the control animals, which received this dietary without any addition of phosphorus. It will be noted in

Fig. 2.—Photomicrograph of epiphysis of figure 1. To the left, rachitic zone of preparatory cartilage, bordered on the right by the transverse band of bone (the phosphorus layer).

table 1, which reproduces the data of this part of the investigation, that no animal failed to show rickets on microscopic examination, and that only one developed lesions of slight intensity. The gross examination of the epiphyses, as well as the roentgenograms, coincide with the interpretation of the microscopic changes. The inorganic phosphorus content of the blood was decreased rather than increased as the result of adding phosphorus to the dietary; for example, in two experiments in which this dietary was employed with an addition of phosphorus, the percentages of inorganic phosphorus were only 1.58 and 2.01 mg. per hundred cubic centimeters. The general condition of the animals and their respective weights seemed uninfluenced by the supplement of

phosphorus, nor was it found to produce any definite changes in the blood picture—in the hemoglobin or in the number of red cells. It may be mentioned, in passing, that difficulty was experienced in obtaining sufficient blood from the vessels of some of these animals, an aspect which will be made the object of further study.

TABLE 1.—*Effect of Adding Elementary Phosphorus to a Rickets-Producing Diet*

Rat	Weight, Gm.	Rickets-Producing Diet	Supplement to Diet (Daily)	Roentgenograms: Rickets	Roentgenograms: Subepiphyseal Band	Rickets: Macroscopic	Rickets: Microscopic	Inorganic Blood Phosphorus, Mg. %
7341*	40-50	Diet 84†	Elementary phosphorus 0.3 mg.	Moderate	Moderate‡	Moderate	Moderate	1.58
7386	64-64	Diet 84	Elementary phosphorus 0.2 mg.	Moderate	Slight‡	Moderate	Moderate	3.44
7387	54-54			Moderate	Moderate‡	Moderate	Moderate	
7492	60-70	Diet 84	Elementary phosphorus 0.2 mg. (14 days)	Marked	Slight‡	Marked	Marked	
7493	40-36			Slight	None	.....	.....	
7494§	46-40			Moderate	Moderate‡	Moderate	Moderate	
7495	34-40		Elementary phosphorus 0.3 mg. (14 days)	Moderate	Moderate‡	Moderate	Moderate	
7687	60-66			Moderate	Slight‡	Moderate	Moderate	
7689	50-52			Moderate	None‡	Moderate	Moderate	
7690	50-49			Slight	None	Slight	Slight	
10221§	64-74	Diet 84	Elementary phosphorus 0.04 mg.	Moderate	None	Slight	Moderate	2.01
10222	44-50			Moderate	None‡	Slight	Moderate	
10223	46-52			Moderate	Moderate‡	Moderate	Moderate	
10224§	42-44			Moderate	None‡	Moderate	Moderate	
10225	60-68	Diet 84	Elementary phosphorus 0.007 mg.	Moderate	None	Moderate	Moderate	
10226	42-46			Slight	None‡	Moderate	Moderate	
10228	40-48			Slight	Moderate‡	Moderate	Moderate	
10227	50-50			None	None‡	Slight	Moderate	
10233	44-44	Diet 84	.....	Slight	None	Marked	Marked	
10234	56-54			None	None	Slight	Slight	
10236	60-60			Moderate	None	Moderate	Moderate	

* Six of this group died.
† Flour 95, calcium lactate 2.9, sodium chloride 2.0, ferric citrate 0.1 per cent.
‡ Subepiphyseal band noted microscopically.
§ Fracture.

As elementary phosphorus did not prevent the development of rickets when the standard 84 dietary was employed, it seemed advisable to make use of a ration which induced rickets of less intensity. Accordingly in one series of tests a dietary was fed which, for some years, has been found of value in our investigation of rickets. In this ration, 5 per cent of the wheat flour of dietary 84 is replaced by an equivalent percentage of dry milk. The milk is a preparation which has been partially skimmed and dried by the roller process. This "5 per

cent dry milk dietary" is, from a nutritional standpoint, far more complete than the 84 diet; it likewise invariably induces rickets. It is not to be recommended for curative experiments, in which the result is gaged by the presence or absence of healing, for calicification or healing not infrequently goes hand in hand with the development of the rachitic lesions. When this diet was employed in our preventive tests, moderate rickets developed in every animal and was as usual associated with the formation of a subepiphyseal band (table 2).

TABLE 2.—*Effect of Adding Elementary Phosphorus to Various Rickets-Producing Diets*

Rat	Weight, Gm.	Rickets-Producing Diet	Supplement to Diet (Daily)	Roentgenograms: Rickets	Roentgenograms: Subepiphyseal Band	Rickets: Macroscopic	Rickets: Microscopic	Inorganic Blood Phosphorus, Mg. %
10138	40-56	5% dry milk	Phosphorus 0.2 mg.	Moderate	Slight*	Moderate	Moderate	
10139	30-43	95% diet 84		Moderate	Moderate*	Moderate	Moderate	
10140	36-54			Moderate	Moderate*	Moderate	Moderate	4.06
10141	40-64			Moderate	Slight*	Moderate	Moderate	
10039	32-60	20% dry milk	.....	Moderate	Moderate	Moderate	Slight	
10040	40-60	80% diet 84		Moderate	Moderate*	Moderate	Slight	
10041	32-50			Moderate	Moderate	Moderate	Slight	2.07
10042	34-56			Moderate	Moderate	Moderate	Slight	
10110	40-60	20% dry milk 80% diet 84	.....	Moderate	None	Marked	Moderate	3.55
10136	34-49			Moderate	None	Slight	Moderate	
10137	44-79			Slight	None	Slight	Slight	
10154	34-40	Diet 84	Phosphorus 0.07 mg. cod liver oil 3 n.g.†	Slight	Moderate*	Very slight	Slight	
10155	50-56			Slight	Marked*	Very slight	Slight	5.72
10156	44-45			Slight (?)	Marked	None	None	
10157	34-42	Diet 84	Cod liver oil 3 Mg.†	Slight	None	None	Slight	
10158	40-44			None	None	None	None	5.12
10159	30-40			Slight (?)	None	None	None	
10150	34-40	Diet 84	.....	Slight	None	Slight	Moderate	
10151	44-50			Very slight	None	Slight	Slight	2.95
10152	50-60			Slight	None	Moderate	Moderate	
10153	46-49			Moderate	None	Moderate	Moderate	
10332§	70-64	Diet 85C‡	Phosphorus 0.2 mg.	None	Slight*	None (?)	Very slight	
10333	61-70	low calcium		None	Slight*	None (?)	Slight	3.58
10334	94-80			None	Slight*	None (?)	Very slight	
10335	80-70	Diet 85C‡	.....	None	None	None (?)	Slight	
10337	60-68	low calcium		None	None	None (?)	None	5.10
10338§	90-85			None	None	None (?)	Very slight	

* Subepiphyseal band noted microscopically.

† This amount of cod liver oil is insufficient to afford protection.

‡ In this dietary potassium phosphate (basic) is substituted for the equivalent amount of calicum lactate in diet 84.

§ Fracture.

When 20 per cent of the flour of dietary 84 is replaced by dry milk, a mild degree of rickets generally develops. The employment of a diet of this kind has the advantage of simulating as closely as possible the dietary conditions which obtain in infant feeding—it furnishes a large amount of milk and is adequate in its content of the various vitamins and dynamic factors. As may be noted in table 2, animals grow well on this ration; it brings about the prototype of what may be termed the plethoric rather than the atrophic type of rickets. On this diet, as in the two diets previously tested, little or no difference could be observed between the rats which had received and those which had not

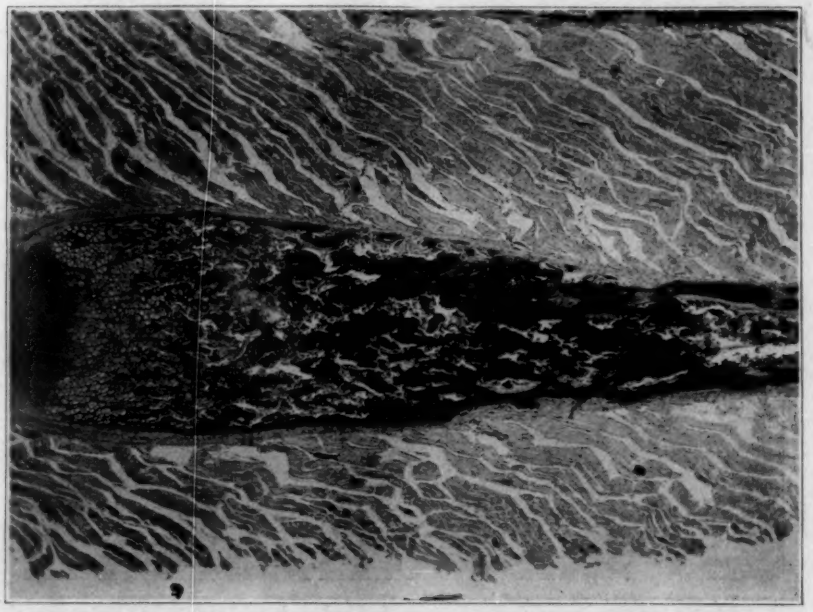

Fig. 3.—Photomicrograph of epiphysis of rat which received rickets-producing diet containing 20 per cent of dried milk and, in addition, elementary phosphorus. Development of rickets and of "phosphorus band."

received the supplement of elementary phosphorus. In every instance rickets developed (fig. 3). The inorganic phosphorus of the blood was higher in the group which was given no phosphorus.

As in every instance the animals had developed rickets on these experimental dietaries, it seemed of interest to ascertain whether if a very small amount of cod liver oil were given in addition to dietary 84, conditions could be rendered so favorable that a supplement of elementary phosphorus would protect the animals from rickets. The addition of cod liver oil seemed especially pertinent, as a mixture of cod liver oil with phosphorus is the preparation originally recommended

by Kassowitz and one which has been employed widely for many years in clinical pediatrics. It seemed possible that the association of cod liver oil with phosphorus might cause it to acquire exceptional antirachitic properties. The average protective dose of cod liver oil for rats receiving diet 84 has been found to be about 7 to 8 mg. a day. Such being the case, about 3 mg. of cod liver oil was fed to each animal, in addition to a supplement of 0.2 mg. of elementary phosphorus. Even under these favorable conditions—the most advantageous which could be formulated—phosphorus was unable to avert the development of rickets, and no distinction could be noted between those animals which received a small

Fig. 4.—Photomicrograph of epiphysis of rat which received the standard rickets-producing diet plus 3 mg. of cod liver oil as well as elementary phosphorus. Development of rickets and of "phosphorus band."

supplement of the cod liver oil, and others which received phosphorus as well as this oil (fig. 4). In both groups the calcification was better naturally than when no cod liver oil was added, and the percentage of inorganic phosphorus in the blood was higher, but the occurrence of rickets was not prevented. It seems evident that if phosphorus is unable to prevent the development of rickets when fortified with a small amount of cod liver oil, it can possess no antirachitic potency.

Having ascertained that phosphorus shows no protective qualities when a rickets-producing diet which is low in phosphorus is fed, its activity was tested in association with a dietary low in calcium. It seemed as if it might be effective when the calcium was low, as Kassowitz

always laid special emphasis on the fact that phosphorus was of value in combating the laryngospasm of rickets, which, as is well known, is a symptom of tetany—a clinical condition which is characterized by a deficiency of calcium. For this purpose dietary 85 C was employed, in which 2.9 per cent basic potassium phosphate is substituted for the equivalent percentage of calcium lactate of the low phosphorus dietary 84. This ratio leads to rickets of a somewhat different character than that brought about by low phosphorus dietaries; the zone of preparatory calcification of the cartilage is not exceptionally wide, although the osteoid tissue is present in excess. The roentgenographic picture is, therefore, frequently negative. As will be noted from an examination of table 2, all the rats which received phosphorus in addition to this dietary developed rickets as judged by the microscopic picture. The degree of rickets was practically the same as among those animals which received the diet unsupplemented by phosphorus. The "subepiphyseal band" developed under these conditions as in the previous experiments, but was somewhat less marked. On account of the defective osteogenesis which is characteristic of the epiphyses of animals fed on this dietary, the formation of the trabeculae which composed the "band" produced a microscopic picture which caused it to resemble the normal. In view of the fact that this diet contains an excess of inorganic phosphorus, we were surprised to find that the addition of elementary phosphorus led to a diminution of the inorganic phosphorus in the blood—3.58 mg. in those animals receiving phosphorus, compared to 5.10 mg. in those receiving nothing but the low calcium ration.

Wegner stated that phosphorus might induce rather than prevent rickets, and some of our microscopic preparations suggested that such might be the case. This aspect was not investigated fully, but a series of rats was placed on dietary 85, which is similar in composition to the rickets-producing diet 84, except that it contains sufficient inorganic phosphate to protect against the development of rickets. Rats fed this diet, with 0.2 mg. of elementary phosphorus in addition, did not become rachitic. It cannot be stated, however, whether the result would have been the same had a borderline or minimal protective diet been given.

COMMENT

It is evident that elementary phosphorus is unable to prevent the development of rickets in rats. There is no reason for believing that its action is essentially different in regard to the rickets of infants, and the statement made about a year ago in reply to the suggestion that elementary phosphorus be employed in the treatment of infantile rickets would seem to hold good: "Recently, we have made careful biologic tests of this kind, and found that phosphorus is of no value in the prevention of rickets. The substitution of phosphorus for other

specific antirachitic agents—ultraviolet or cod liver oil—would be a decided step backward, and is fraught with danger to one of the most promising fields in preventive medicine." [9]

The favorable results which have been reported by others have been interpretations of laboratory investigations as well as of clinical observations. In both, the fundamental error has been made of regarding rickets as a disorder characterized by a simple lack of calcification and, therefore, of drawing the deduction that any factor which is capable of stimulating calcification must be, through the virtue of this quality, a potent antirachitic agent.[10] The failure of elementary phosphorus to bring about protection against rickets emphasizes once more the essential nature of this disorder—the fact that its lesions do not come about merely from a lack of deposition of inorganic salts in the epiphyses. It is true that elementary phosphorus is able to stimulate calcification at the epiphyses, but as it is unable to calcify the preparatory cartilage or osteoid tissue, it is impotent in preventing the advance of the essential pathologic process. Nor have we been able to convince ourselves that it is of definite value in craniotabes. In "nonrachitic softening of the ribs," [11] it was given a thorough trial without success. When we bear in mind its narrow and circumscribed field of ossification, these failures are not to be wondered at.

The stimulating action of elementary phosphorus on bone formation ("subepiphyseal band") and lack of action in relation to the cartilage, which lies immediately adjacent, furnishes an excellent illustration of the specific action of a chemical substance for certain tissues. How the ossifying process is brought about by the phosphorus must remain a matter of conjecture for the present. The original explanation of Wegner, attributing it to an "osteoplastic irritation," seems plausible; according to this theory the irritative or toxic properties of the phosphorus lead to the production of the bony trabeculae. Kassowitz did not believe that an increase in the formation of bone took place, but that the band resulted rather from a lack of absorption of bone. Furthermore, how it comes to pass that osseous tissue is produced only at this site and nowhere else within the shafts of the bones, is a problem which must await further investigation.

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9. Hess, A. F.: Irradiated Foods, Radium and Phosphorus, J. A. M. A. **85**:458 (Aug. 8) 1925.

10. Some years ago, Schabad carried out a metabolism experiment which seemed to show that phosphorus given in addition to cod liver oil brought about an increase in the retention of calcium. Such may be the case, but this does not signify that the retained calcium is deposited in the rachitic zones. The "phosphorus bands" and the calcification along the periosteum must result in a retention of calcium.

11. Hess, A. F.: Nonrachitic Softening of the Ribs in Infants and Children, Am. J. Dis. Child. **28**:568 (Nov.) 1924.

It has been demonstrated by various authors, more recently by Phemister and his co-workers, not only that transverse bands of bony tissue can be brought about by feeding phosphorus, but that if this substance is alternately given and discontinued, a series of dense, parallel bands is produced, separated by intervening areas of normal density. This leads one to inquire whether a pathogenetic connection exists between the transverse bands which come about as a result of feeding elementary phosphorus, and the "transverse lines" which have been noted and described so frequently in the epiphyses of infants. As is well known, one or more of these transverse lines is frequently seen by

Fig. 5.—Photomicrograph of tibia of rat which received the standard rickets-producing diet plus 0.007 mg. daily of elementary phosphorus. Rickets, a "phosphorus band," and an increase of periosteal bone.

means of the roentgen rays or on microscopic examination somewhat distal to the epiphyseal junctions. These horizontal lines of increased calcification are attributed generally to remissions in the course of the rachitic process. Some years ago, in the course of a study of the roentgenologic appearance of the epiphyses of a large series of infants who were followed month by month in a childcaring institution, transverse lines frequently were found to have developed under observation. Although there had been no change in the diet or in the general hygienic surroundings of the infants, this phenomenon occurred not only in the spring and summer when it might have been attributed to seasonal

influences, but also during the winter months. The explanation of the development of these "transverse lines" has always been lacking. In view of these experiments with elementary phosphorus, the question arises as to whether some of them may not be occasioned by irritative substances similar in physiologic action to elementary phosphorus.

Attention should be called to the minuteness of the amount of phosphorus which sufficed to bring about the calcification bands at the epiphyses. As stated, the usual dose which was given to each animal was 0.2 mg. daily. It may be noted in table 1 that these roentgenologic and histologic changes were occasioned by 0.04 and even by 0.007 mg. (fig. 5). These are amounts which call to mind the action of iodine in relation to the prevention of goiter and the potency of some of the bacterial poisons. It is clear that in our concept of agents which can bring about specific tissue changes, we shall have to think in terms of minute fractions of a milligram.

CONCLUSIONS

The feeding of small amounts of elementary phosphorus to rats leads to the production of "a phosphorus band" at the epiphyses of the long bones. This consists of a transverse layer of dense compact osseous tissue, lying transversely, immediately adjacent to the proliferating cartilage. It is clearly visible on gross examination when the head of the bone is sectioned longitudinally, and stands out prominently in the roentgenographic picture. On microscopic examination, the band is found to consist of heavy trabeculae of bone, and to be limited to the subepiphyseal region of the shaft. As little as 0.007 mg. of phosphorus a day sufficed to bring about this change in the epiphyses.

Elementary phosphorus, whether given in small or in large doses, was unable to prevent the occurrence of rickets to any degree whatsoever. Even when the diet was but slightly rachitogenic, the addition of phosphorus did not protect against rickets. Furthermore, combining phosphorus with an inadequate amount of cod liver oil failed to render the oil adequate. These failures in protective action were noted on diets low in phosphorus as well as on a diet low in calcium.

A marked increase in calcification of the long bones (subepiphyseal band) took place concurrently with the development of typical rachitic lesions. That such is the case indicates that rickets must be regarded not as the result of a failure of calcification, but as a disorder characterized by specific changes in the preparatory cartilage and in the growing bone. Elementary phosphorus fails because it is unable to bring about calcification within this cartilage or to prevent the undue formation of osteoid tissue.

TOPOGRAPHY OF INTERLOBAR PLEURISY IN INFANTS BY LIPIODOL INJECTION

LOCALIZATION BY THE USE OF LIPIODOL OF INTERLOBAR PLEURISY MASKED BY MASSIVE PNEUMONIA *

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A certain number of authors have already drawn attention to the association between interlobar pleurisy and massive pneumonia. Thiroloix and Rosenthal [1] consider massive pneumonia to be the initial reaction of an interlobar pleurisy. Chauffard, and later his pupils, Guy, Laroche, Boidin and Huber, have showed the inadequacy of radioscopic examination in the diagnosis of certain interlobar pleurisies.[2] More recently, Ardin-Delteil and Raynaud,[3] Caussade and Deberdt [4] have cited observations of interlobar pleurisy masked by massive pneumonia. This question has been the subject of a thesis by Pesle (Algiers, 1922). Finally, Ardin, Delteil, Derrien, Lévy and Valensi, from a review of the reported cases, have made the following conclusions:

1. That massive pneumonia may lead to an encysted pleurisy, and that one may consider it as a pulmonary reaction vis-à-vis a localized pleural inflammation.

2. That the diagnosis may be uncertain for a long time, even until the contents of the abscess cavity are expectorated, without radioscopy being of decisive aid.

A recent case of similar nature, which came under our observation, presents the following history:

REPORT OF CASE

A child, aged 2 years, entered our service at the Hôpital Hérold, March 8, 1926. The weight on admission was 9.9 Kg. and the temperature 102 F. Functional and physical signs of consolidation of the upper two-thirds of the left lung were present. The tuberculin reaction was negative. The presence of absolute flatness under the clavicle and over the upper half of the back caused us to make an exploratory puncture, March 10. The point of puncture was somewhat median to the angle of the scapula, but nothing was obtained.

March 11, fluoroscopy showed a total and homogeneous opacity of the left hemithorax. A diagnosis of massive pneumonia was made, the roentgenogram having confirmed the fluoroscopic picture. We felt, in spite of the absence of the

* Received for publication, June 15, 1926.

1. Thiroloix and Rosenthal: Bull. et mém. Soc. méd. de hôp. de Paris, July, 1901.

2. Chauffard, A.: Ann. méd., June, 1914.

3. Ardin-Delteil, P., and Raynaud, M.: Paris méd. **10**:487 (June) 1920.

4. Caussade, G., and Deberdt, P.: Bull. et mém. Soc. méd. de hôp. de Paris **45**:1635 (Dec. 9) 1921.

tubercle bacillus from stomach washings, that the infection was tuberculous, so decided to try artificial pneumothorax. The temperature at this time was 100 F.

Four days later, in introducing the trocar of Küss into the pleural cavity in the midaxillary line, a small amount of serous fluid was found and the idea of collapsing the lung was given up. No further fluid was obtained from daily chest punctures on three consecutive days.

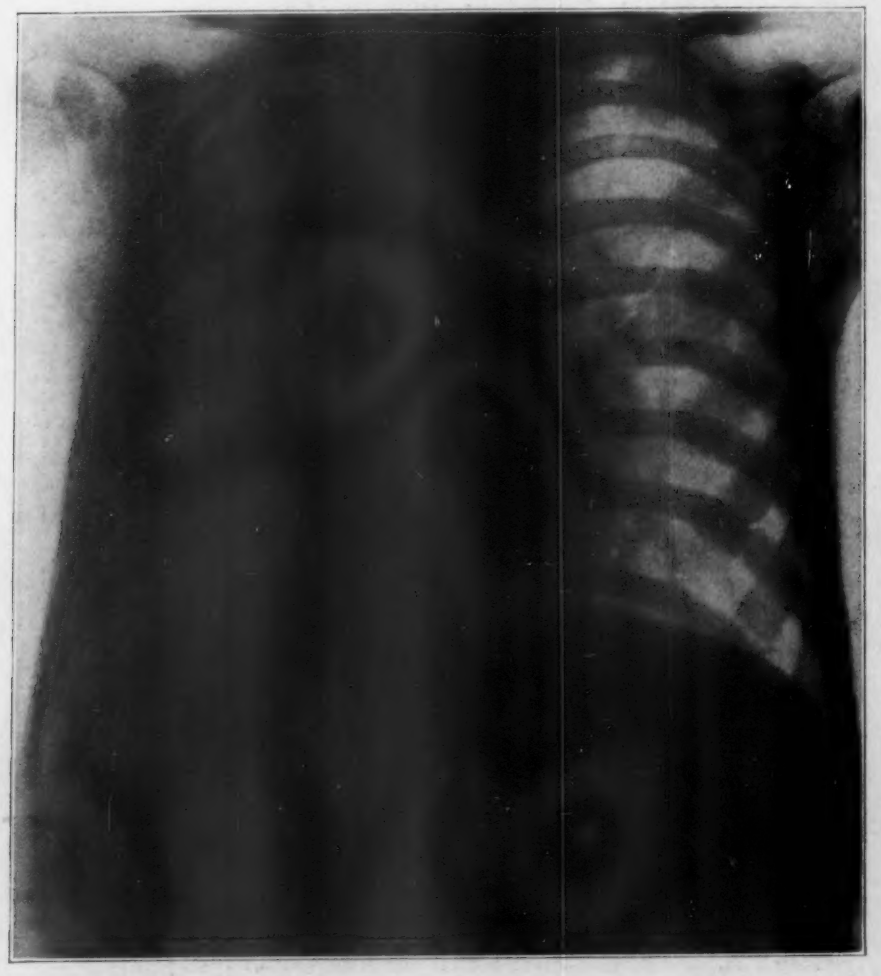

Fig. 1.—View of thorax before injection when first observed.

March 19, the temperature had risen to 102 F., and a puncture made in the midaxillary line gave a greenish pus containing numerous pneumococci. Radioscopic examination performed the same day showed no change in the lung shadow.

March 22, the patient had lost 1 Kg. in weight and the temperature was 100 F. A puncture made to evacuate the cavity produced 50 cc. of pus. Radioscopic examination, performed after the puncture, still showed no change in the shadow. A diagnosis was made of encysted pleurisy, but exact localization of this was made impossible on account of the opacity of the roentgenograms of the pneumonic process.

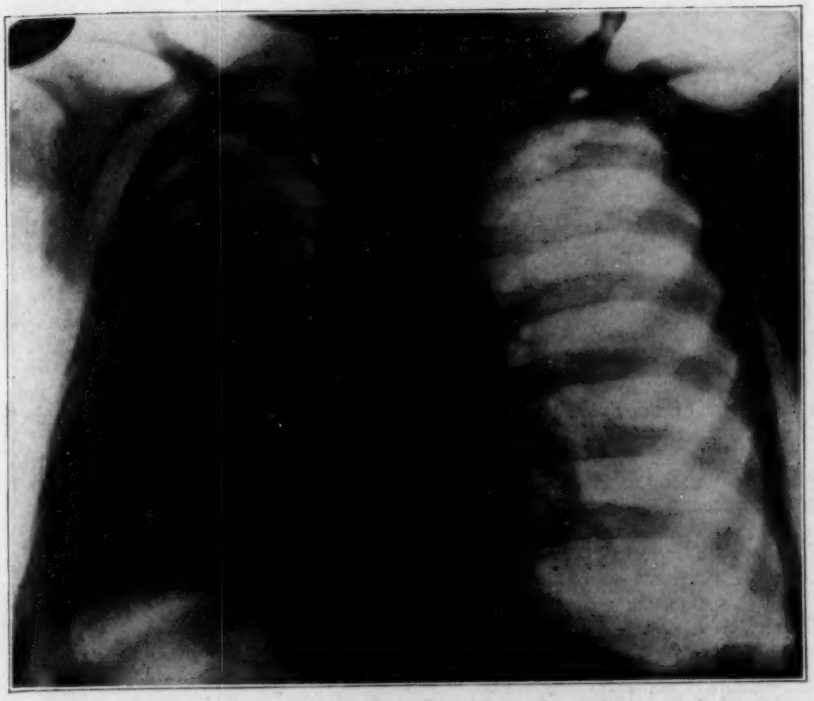

Fig. 2.—Anteroposterior view after lipiodol injection.

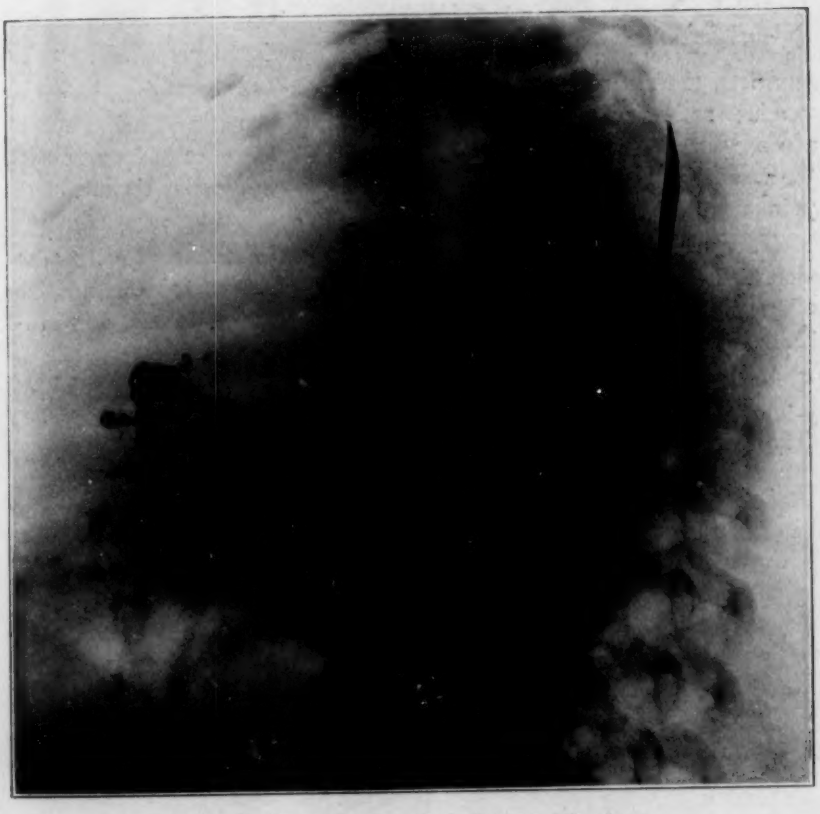

Fig. 3.—Lateral view after lipiodol injection.

On April 3, with a persistent "pump-handle" temperature of 104 F., and with continued loss of weight, a new puncture was made for drainage, 15 cc. of pus being obtained. This was replaced by 15 cc. of tepid lipiodol. A roentgenogram was taken immediately after and these plates are shown here. These plates permit the localization of the cavity, filled with lipiodol, in the antero-inferior part of the left interlobar fissure. The shadow of the lipiodol seen from the front reaches almost to the level of the diaphragm. From the profile it presents a generally oblique direction extending from the back forward without reaching the postero-superior limit of the interlobe, but by changing the position of the patient, which is easy with a child, the shadow of the lipiodol can be displaced throughout the extent of the fissure.

The temperature remained at 98.5 F. from April 9 to 17. At the latter date the shadow of the lipiodol remained, standing out on the less intense shadow of the pneumonic infiltration.

April 21, after another rise of temperature to 104 F., another evacuating puncture was made and the pus withdrawn contained lipiodol and, on culture, pneumococcus.

April 27, there was another rise of temperature and the weight, which had fallen to 8.7 Kg., had increased to 8.9 Kg. The temperature for two days had been 98.5 F.

In this case, we applied the method of Sicard and Forestier of injections of lipiodol, as advised by Noël and Fiessinger and André Lemaire.[5]

It is not surprising to see the shadow of the lipiodol stand out clearly against the shadow of the massive pneumonia. One of us has already shown, with Robert Lévy and Julien Marie, by intratracheal injections of lipiodol, bronchial dilatations hidden by a lobe of massive pneumonia.

CONCLUSIONS

The study of this case permits of the following conclusions:

First, the association between encysted pleurisy and massive pneumonia; further, we have been able to realize, for the first time, it seems, a process which permits us, as Fiessinger and Lemaire have proposed, "to visualize and outline surgically, without fear of error, certain intrapulmonary collections."

This procedure may be used even after the formation of a bronchopleural fistula, since the tract of the fistula is, in general, too small to allow the penetration of lipiodol, and its evacuation by the bronchial route.

It should be added that not only is the procedure harmless, as we supposed from our work on intratracheal injections of lipiodol, but a definite improvement was noted following its use, both in the temperature curve and in the weight.

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5. Fiessinger, N., and Lemaire, A.: Presse méd. **34**:209 (Feb. 17) 1926.

PULMONARY ACTINOMYCOSIS

REPORT OF A CASE IN A BOY AGED FIVE YEARS *

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AND
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PHILADELPHIA

In a recent publication, Sanford and Voelker [1] report the protocols of 670 cases of actinomycosis occurring in the United States. This is by far the most comprehensive survey ever made of this disease. Analysis of their cases shows that the condition occurred forty-two times in children under 15 years of age. Of this group, there were only five cases of primary pulmonary infection. The disease also is very rare in the states of the Atlantic seaboard. We, therefore, feel justified in reporting another case of pulmonary antinomycosis, especially since it occurred in a child, aged 5 years, who had lived all of his life in a large eastern city.

CASE HISTORY

L. C., a boy, aged 5 years, was admitted to the Children's Hospital, Nov. 22, 1925, with the chief complaint of pain in the left side of the chest.

Present Illness.—The boy had had a slight cough ever since an attack of "pneumonia" in June, 1925. The onset of the present condition was November 5, when he developed a sharp pain in the left side of the chest; this had diminished but was still present on his admission to hospital. Other symptoms were a loose, productive cough without hemoptysis, irregular temperature in the evening, and night sweats with loss of weight and strength.

Past Medical History.—The boy had had measles and varicella, and pneumonia (?) in June, 1925. He had had frequent attacks of tonsillitis and pharyngitis, and tonsillectomy and adenoidectomy had been performed in 1924. Otherwise the history was negative.

The family history was essentially negative for all chronic diseases. There was no history of syphilis or tuberculosis. The patient's father operates a small refreshment stand at a suburban college, but is a barber by trade. The home conditions, hygiene and neighborhood are good.

The boy had been breast fed for twelve months, after which he was given cow's milk and a general diet. His dentition was normal and he sat up, walked and talked at the usual times.

Physical Examination.—The patient weighed 35 pounds (16 Kg.); the temperature was 100 F.; pulse, 134; respirations, 40. He was undernourished and pallid, mentally alert, and not acutely ill. Rachitic deformities of the extremities and ribs were seen. There were no dyspnea, cyanosis or clubbed fingers. The only other positive findings were in the chest.

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* Received for publication, June 15, 1926.

* From the medical service, Children's Hospital.

* Read by title before the American Pediatric Society, Niagara Falls, Canada, June 2, 1926.

1. Sanford, A. H., and Voelker, M.: Actinomycosis in United States, Arch. Surg. **11**:819-841 (Dec.) 1925.

The chest showed limitation of expansion, and slight fulness of the interspaces on the left side, with dulness to percussion and increased voice transmission but absent tactile fremitus. Over the left base, distant bronchial breathing was heard with occasional showers of crackling râles. In the second interspace midway between the sternum and the axilla, there was a tender, slightly "boggy" swelling, suggestive of an "empyema necessitans" in an unusual situation. A hyperresonant note and puerile breath sounds were heard over the right lung. The heart was displaced slightly to the right, but the sounds were of good quality. No murmurs or friction sounds were heard. The urine was normal; the blood showed a slight secondary anemia with 23,000 white blood cells and 89 per cent polymorphonuclear

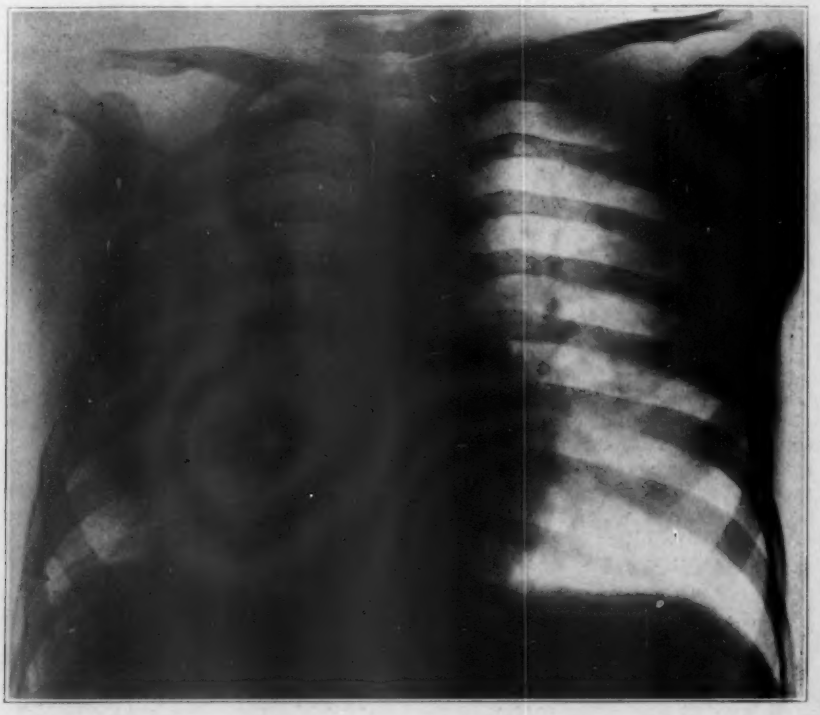

Fig. 1.—Roentgenogram of pulmonary actinomycosis, taken on Nov. 4, 1925.

cells. An intradermal tuberculin test with 0.1 mg. of old tuberculin was negative. The sputum was negative for tubercle bacilli and the Wassermann examination of the blood was negative.

The roentgen-ray examination on November 23 showed that the left side had a dense shadow occupying the entire side of the chest. The heart was displaced to the right, which was probably due to a massive collection of fluid or pus. The patient was then transferred to the surgical service of Dr. W. Estell Lee (later succeeded by Dr. John Speese). The temperature continued to be slightly elevated in the evening; there were night sweats, with a productive cough and moderate leukocytosis. The first operation was performed by Dr. Lee over the area of swelling in the second interspace. A few drops of pus were obtained outside of the pleural cavity, and the pleura was found to be thickened and densely adherent but no other area of suppuration could be found. Smears and cultures of the pus

were sterile. Following the operation, repeated examinations of the sputum were reported negative for tubercle bacilli and fungi.

The second operation was performed in the midaxilla, sixth interspace. This disclosed the same thickened, almost cartilaginous condition of the pleura, but without any collection of pus. Both wounds failed to heal promptly and continued to drain grayish pus. Repeated roentgenograms of the chest showed extension of the process, with marked cardiac displacement.

In February, the patient developed several blue, fluctuating, tender swellings over the left thoracic wall. These were incised, and thick yellow pus was obtained, cultures and smears of which proved to be negative. These wounds also did not

Fig. 2.—Roentgenogram of chest taken on March 16, 1926, showing increased density and displacement of the heart, with extension to the opposite lung.

heal, but continued as sinuses with indurated puckered margins. The temperature was unchanged, but the boy's general condition improved with a good diet, cod liver oil and ultraviolet therapy.

All of the sinuses were then discovered to be communicating, and on March 1, some pus taken from one of them showed organisms resembling the mycotic group, which were not characteristic in smear or culture. Since the patient had developed several more large thoracic abscesses, a more extensive exploratory thoracotomy was performed by Dr. Speese on March 5. Abscesses were opened and thick pus was evacuated. The chest wall, ribs and pleura were necrotic and infiltrated; specimens were removed for diagnosis. The pleura was densely adherent at all points and the lung was apparently solid. Examination of this pus showed the typical "sulphur granules," and mycelial organisms of the "ray fungus"—*Actinomyces bovis*. Sections of the pleura also showed *Actinomyces*.

Following treatment for operative shock and the use of transfusion of citrated blood, the patient improved slightly. The wounds were packed daily with gauze and irrigated with 1:2,000 solution of oxyquinolin sulphate (chinosol). These cavities filled in nicely with dense granulation tissue. The boy was given a nourishing diet and potassium iodide in ascending doses until he was taking 30 grains per day. Following operation, his temperature was always higher than before, and subject to greater remissions, and he showed leukocytosis. No new abscesses formed.

On March 29, the patient developed a severe pharyngitis with cervical adenitis which prostrated him. His temperature in the evening rose to 107 F. On the morning of March 30 he was given 45 mg. of gentian violet in a 0.5 per cent

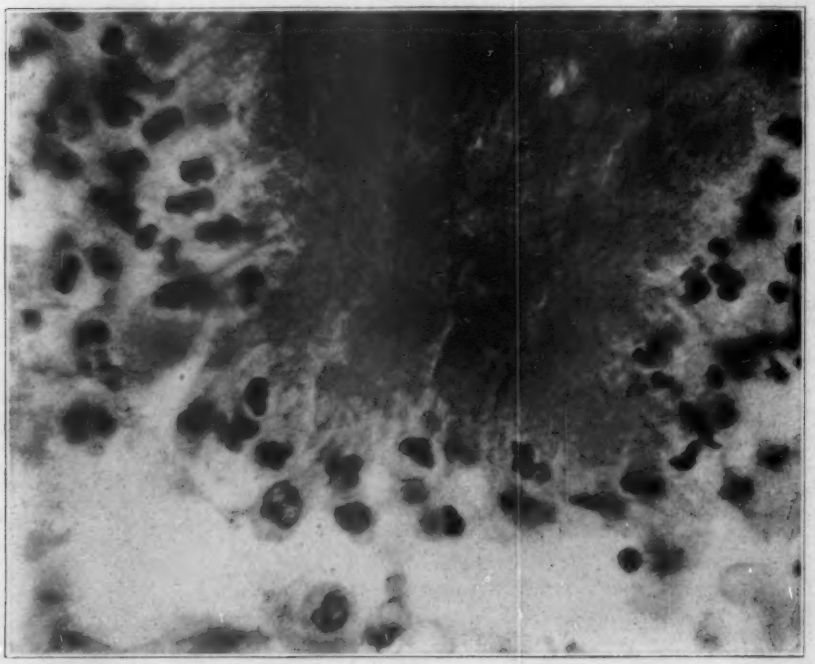

Fig. 3.—Actinomyocis bovis of the pleura, showing the "ray fungus" with the clubbed ends (× 1004).

solution, intravenously. No untoward effects were noted. March 31, he was much worse and signs of a bronchopneumonia at the right base were elicited. A roentgenogram showed diffuse mottling through that lung. He was given a transfusion with 80 cc. of citrated blood, though almost moribund, but he died about four hours later. Necropsy was not permitted.

COMMENT

Human actinomycosis is a comparatively rare disease. The spores of the organism are widely distributed in nature and are blown about freely by the winds. There is, however, high natural resistance in man and in most animals to infection. Cattle are least immune, and frequently develop lesions on the tongue and jaws. This gives rise to

"lumpy jaw." More rarely the disease occurs in dogs, horses, cats, sheep and goats.

It is said that the human infections usually follow the practice of chewing straw, or picking the teeth with straw or the shafts of grain. Infection from the milk or flesh of cattle has never been proved. The spores are not resistant to high temperature, and would be killed by pasteurization—to which practically all of the Philadelphia milk supply is subjected.

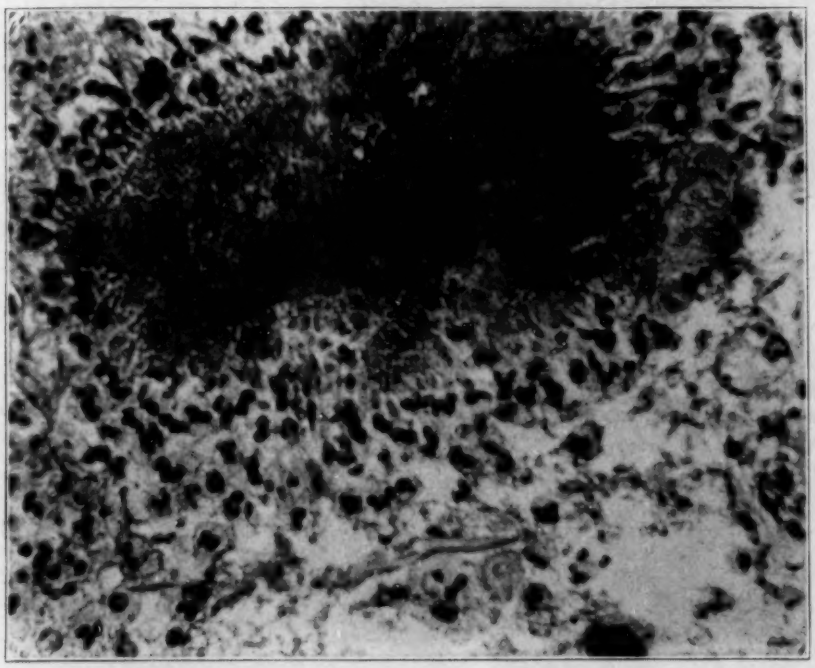

Fig. 4.—Actinomycosis bovis of the pleura (× 736).

This child was born and raised in Philadelphia, and his visits out of the city were limited to occasional vacations of short duration at Atlantic City, a seashore resort. He was never exposed to the possibility of rural infection, nor to the fruit or other wares at his father's store. The home was well kept and was not situated near stables or abattoirs.

The family did have several cats as pets. One of these cats died mysteriously about June, 1925. Necropsy was not performed on it. The father reported that the cat was sick only twenty-four hours before death.

The disease in this case probably had its inception at the time of the illness in June, 1925, diagnosed as pneumonia, and from which the

patient never fully recovered. The disease at the time of admission in November was well advanced, and again simulated a pneumonic process with fluid in the chest. Delay in diagnosis was due to the fact that no organisms were ever found in the sputum, and were not identified in the pus until March, through the condition was suspected before this time.

Differential diagnosis of these cases is at all times difficult. It must be differentiated from chronic fibroid phthisis, diffuse pulmonary neoplasms, empyema, or chronic pneumonia with pleurisy. Diagnosis can be established only on the finding of the organisms in the sputum, or in the pus from the thoracic sinuses which usually form; these have a characteristic puckered, indurated and grayish appearance. The pus is composed of necrotic tissue, and cellular detritus with scarcely any leukocytes. The skin and ribs are necrotic, but are not definitely invaded by the fungus.

Various therapeutic measures are suggested by writers, but a survey of the case reports shows that treatment is usually successful in those infections limited to the jaw or skin. Visceral antinomycosis has a high mortality. Various remedies, iodides, gentian violet intravenously, ultraviolet irradiations and transfusions had no appreciable effect in our case. Surgical measures served only to clear up abscesses and sinuses.

We were in consultation with Dr. V. G. Kimball, assistant professor of veterinary medicine, University of Pennsylvania, who could offer no suggestions concerning etiology or treatment other than those noted.

SUMMARY

1. A case of primary pulmonary actinomycosis occurring in a child, aged 5 years, is presented.

2. Factors relating to the mode of infection in this case are not known, but transmission from a cat is suggested.

PYURIA IN MALFORMATIONS OF THE URINARY TRACT *

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REVIEW OF LITERATURE

In a series of cases of urinary infection in children, which were collected and reported,[1] a baby, aged 6 weeks, was observed who had persistent pyuria with continuous fever, and presented a tumor in the median line which could be palpated through the abdominal wall. On necropsy this tumor proved to be a horseshoe kidney. The large dilated pelvis contained a purulent secretion, and numerous miliary abscesses were found distributed through the kidney parenchyma. This was my first acquaintance with pyuria in malformations of the urinary tract.

Since that time, a number of children with protracted pyuria have presented themselves, and malformation of the bladder, ureters or kidney could be demonstrated in some of them, frequently by cystoscopic examination, occasionally at necropsy.

This etiologic factor as a cause of chronic pyuria has received scant attention in the pediatric literature. Gerstenberger and Wahl,[2] in discussing the predisposing causes of pyelitis, state that malformations of the urinary tract such as hydronephrosis, split ureter, exstrophy of the bladder, valve-like folds in the ureter and urethra and renal calculi are factors in the production of pyuria.

All writers on the subject are agreed that malformations in any portion of the urinary tract which interfere with kidney drainage or with the blood supply, make the urinary organs prone to infection.

Wolfang Courtin [3] has reported three cases of pyuria associated with malformations. In one, the pelvis of the right kidney was enlarged, with the ureter straight but as wide as the finger, demonstrated by a pyelogram. Cystoscopic examination of this child at a later time showed a diverticulum of the bladder near the right ureteral opening, producing the obstructive symptoms referred to. He also reports a boy, aged 2½ years, who had continued pyuria with fever, loss of appetite and loss of weight, with a palpable tumor in the midline of the abdomen the size

* Received for publication, July 22, 1926.

1. Abt, I. A.: Urinary Infection in Children, J. A. M. A. **49**:1972 (Dec. 14) 1907.

2. Gerstenberger, H. J., and Wahl, S. A.: Ohio State M. J. **20**:353 (June) 1924.

3. Courtin, W.: Monatschr. f. Kinderh. **31**:24 (Oct.) 1925.

of a fist. At necropsy the ureters were as thick as a loop of small intestine. The bladder was increased in size with its walls thickened and hypertrophied. On more careful examination it was found that there was a diverticulum of the bladder which caused the ureteral obstruction.

Aschoff,[4] in discussing diverticula of the bladder, points out that the favorite site is the floor of the bladder behind the interureteral ligament. They may also be found at the opening of the ureters or the urachus. If the urine collects in the diverticulum in sufficient quantity, it stagnates and causes infection of the protruding sac. This may lead to pericystitis or even to perforation.

Hyman,[5] in reporting three cases of diverticula of the bladder, found pyuria in all. He remarks that it is a curious fact that one rarely sees these cases early in their course when the urine is clear. It is not until urinary infection intervenes, with concomitant urinary symptoms, that medical advice is sought. All of his subjects showed a residual urine. In one infant, retention was produced by pressure of two large sacs causing obstruction at the neck of the bladder.

Clinton K. Smith,[6] writing on lesions of the ureters, says that congenital malformations are of common occurrence and that they are often the cause of diseases of the upper urinary tract. They may produce hydronephrosis, pyelitis, pyelonephritis or nephrosis.

Obstruction produces stasis, and dilatation invariably follows unrelieved obstruction. It may be considered that stasis is the prime factor in the development of urinary infection.

Dilatation of the ureter and bladder may at times be due to spinal cord lesions. Braasch[7] points out that the disturbance of innervation may affect not only the bladder but also the ureters, and Ransohoff[8] says that dilatation of the bladder and ureters, dependent on spinal cord lesions, may occur at any period of life from infancy to extreme old age.

It is the purpose of this paper to point out that malformations of the urinary tract cause stasis, and when stasis occurs infection ensues. The obstruction may be congenital, as in anomalies of the urethra or the meatus, such as epispadias, hypospadias and phimosis, or of the bladder, such as diverticula or exstrophy. It may be caused by strictures or by anomalies of the ureter or congenital malformation of the kidney, or spinal cord lesions. The obstruction may also be caused by acquired lesions in any portion of the urinary tract.

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4. Aschoff: Textbook of Pathological Anatomy **2**, 562.
5. Hyman, A.: Diverticula of the Bladder in Children, Surg. Gynec. Obst. **36**:27 (Jan.) 1923.
6. Smith, C. K.: Surg. Gynec. Obst. **38**:509 (April) 1924.
7. Braasch, W. F.: Dilatation of the Ureter and Renal Pelvis, J. A. M. A. **73**:734 (Sept. 6) 1919.
8. Ransohoff: Keen's Surgery **8**, 507.

The following cases illustrate the various phases of obstruction and infection in malformations of the urinary tract in children. Pyuria was the outstanding manifestation in all instances. Other symptoms complained of were dysuria, vomiting, chills and fever, pallor, suprapubic pain, incontinence of the urine, and frequency of urination.

In all of the cases the pyuria was protracted and did not improve under the usual medical treatment, which was continued in some instances for weeks or months. The true nature of the condition was revealed only by cystoscopic examination or by roentgenograms. In several instances malformations were proved by necropsy.

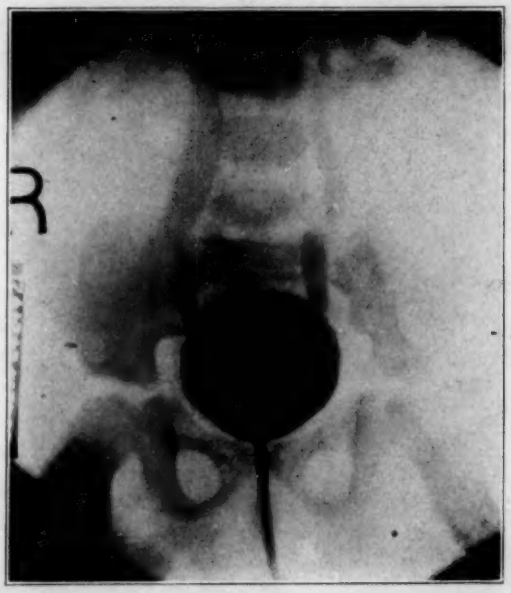

Fig. 1.—Enlarged thickened bladder and dilated ureters.

REPORT OF CASES

CASE 1.—R. A., a girl, aged 8 years, was admitted to the hospital Dec. 4, 1920. She had a history of otitis, malnutrition and headaches. Her family had recently brought her from Russia, where they suffered many hardships. It was noted that the abdomen was enlarged, and her mother reported that this had been so since she was 3 years old. Several days after admission it was noted on ward rounds that the bladder was distended almost to the umbilicus, and the nurse was directed to catheterize her. The urine obtained contained blood, and gave a positive reaction for albumin, and in a few days the urine was found loaded with leukocytes. The child had intermittent febrile attacks, the temperature rising to 101.8 F. at times, and then for a few days would come down to normal only again to be followed by an almost continuous elevation.

On examination by cystoscope on Feb. 22, 1921, by Dr. J. S. Eisenstaedt, the attending urologist, it was observed that there was a pouch-like diverticulum at each ureteral opening. Roentgen-ray examination showed both ureters to be markedly dilated.

Fluid introduced into the bladder easily found its way into the dilated ureters. The child remained in the hospital for nearly a year, with almost continuous fever and persistent pyuria. She became greatly emaciated. The enlarged thickened bladder could be palpated through the abdominal wall.

After this long siege, she was seized with convulsions on December 26. Following this attack she was drowsy, and her face became puffy. The following day she had more convulsions, became dyspneic and died.

The examination of the urinary tract after death showed both ureters greatly dilated, the right more than the left. In their widest portion they would easily admit the index finger. On section of the kidneys, Dr. Oscar T. Schultz reported that the pelves and calices were found greatly dilated and filled with cloudy purulent fluid. On account of the dilatation, the medullary portion of the kidney was greatly reduced in thickness. The ureters were also filled with purulent fluid.

On opening the ureteropelvic junction of each kidney, several horizontal folds were seen, but these did not seem sufficiently large to offer obstruction to the flow

Fig. 2.—Ureters markedly dilated.

of urine. At the distal ends the ureters opened into the bladder through wide orifices without obstruction. The bladder was hypertrophied, and on cut surface was seen to be from 0.5 to 1 cm. thick. The inner surface was trabeculated and hyperemic in some areas.

The histologic examination of the kidneys showed that the glomeruli were granular and necrotic in spots. In the new formed interstitial tissue there were islands of dense lymphoid tissue, though the striking feature of the kidney changes was the disappearance of the secreting tubular parenchyma of the cortex. In brief, the observations were: Bilateral dilatation of the ureters, bilateral pyonephrosis, hypertrophy of the bladder and chronic interstitial nephritis.

The clinical course and pathologic observations would suggest an obstructive lesion interfering with outflow from the bladder. Careful search failed to reveal any narrowing in the urinary tract. Whether the condition was due to a congenital anomaly of the ureters or bladder, whether it was acquired, or whether it was due to a spinal cord lesion, we are unable to say. The fact remains, however, that the persistent pyuria and fever were incidental to the process of dilatation of

the ureters and kidney pelves, with extensive degenerative changes in the kidney itself.

CASE 2.—A. D., a girl, aged 5 years, was admitted Feb. 3, 1921. On entrance to the hospital the child suffered from pain in the suprapubic region, and from painful and increased frequency of urination. In addition she had elevation of temperature, loss of appetite, vomiting and was slightly emaciated.

The usual physical examination revealed nothing definite. There were no palpable masses in the abdomen, though there appeared to be tenderness in both kidney regions. The urethral orifice was hyperemic and swollen.

Pyuria continued undiminished in spite of every form of internal treatment. The patient left the hospital, and returned at intervals without any abatement of

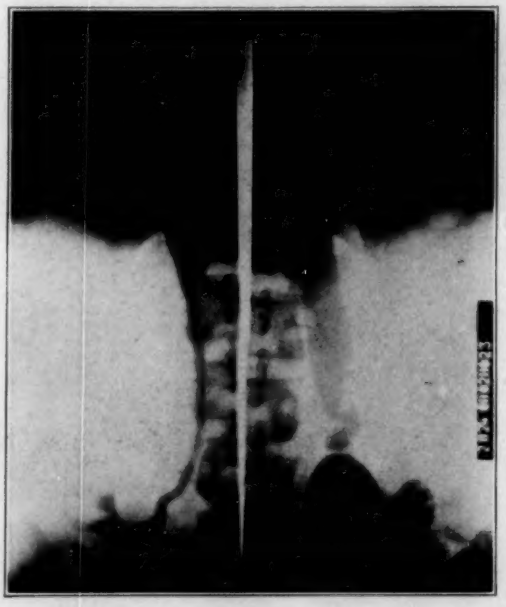

Fig. 3.—Dilatation of both ureters and renal pelves.

symptoms. On Sept. 23, 1923, cystoscopic examination by Dr. Eistenstaedt showed that the bladder was trabeculated. There was no obstruction in the urethra. At the location of the ureteric orifices huge openings were seen symmetrically placed. Roentgenograms of the urogenital tract showed the ureters to be markedly dilated, and at least one-half inch in diameter.

Chills and intermittent fever were of frequent occurrence both at home and during the stay in the hospital.

This child is still living and under treatment. She was brought to the hospital for urologic and general treatment in June, 1926. She was pale and emaciated, and urologic examination did not show any improvement in the condition. Pyuria and fever continue.

She is undoubtedly suffering from a pyelonephritis, with dilatation of the ureters and pelves of the kidneys without any definite evidence as to the nature of the obstructing cause. In the absence of positive obstructive symptoms, the presumption seems justified that the dilatation of the ureters is congenital in origin.

CASE 3.—M. R., a girl, aged 5 years, was admitted to the hospital May 21, 1924. She suffered from pyuria, anorexia and urinary incontinence for three or four years. At the age of 6 months she began to have elevation of temperature. Several months later pus was discovered in the urine. The pyuria has been continuous since that time.

The cystoscopic examination, made by Dr. Irvin Koll, showed that the bladder was trabeculated, and there were apparently diverticula at the urinary orifices. The examination showed the mucosa of the bladder to be thickened and inflamed. Roentgen-ray examination showed a double ureter on the left side, and dilatation of both ureters and the renal pelves.

These observations, with persistent pyuria and fever, suggest a congenital origin of the urinary malformations with subsequent infection of the urinary tract.

CASE 4.—I. D., a boy, aged 4 years, was admitted to hospital Nov. 11, 1925. The chief complaints were frequency of urination, incomplete emptying of the bladder and pyuria, first noticed eight months before admission. The kidneys were not palpable. The bladder could be felt up to the umbilicus, and when

Fig. 4.—Left megalo-ureter and dilated bladder.

distended it showed irregularity of the right half of the vertex; the general direction of the bladder was toward the right.

Cystoscopy and cystograms, made by Dr. Koll, showed that there was a left megalo-ureter with descent of the left kidney.

November 26, suprapubic incision revealed a large distended bladder, extending above the umbilicus. On opening, a valve-like slit was found on the left side which led up to what was evidently a megalo-ureter. A ureteral catheter was introduced and retained in place. A catheter was also introduced into the bladder and the wound closed.

December 2, because of the persistent pyuria, megalo-ureter and infected hydronephrosis, the left kidney and large thickened ureter were removed. The patient improved and was discharged from the hospital, December 23.

It is assumed that the malformation was due to an obstruction of the left ureter at the vesical orifice by a reduplicated fold of mucous membrane. This produced dilatation of the ureter and pelvis of the kidney with subsequent hydronephrosis, leading eventually to an infection of the entire urinary tract on the left side.

CASE 5.—M. S., a girl, aged 2½ years, was seen repeatedly in the office before admission to the hospital. She was first examined Sept. 21, 1925. The mother reported that the child had had "fever" for five weeks, which first occurred after vaccination. Before this she apparently was well.

Fig. 5.—Dilatation of pelvis of right kidney.

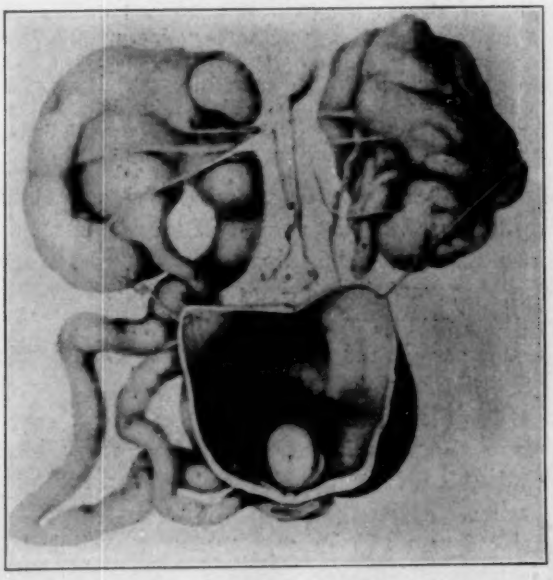

Fig. 6.—Congenital obstruction of right ureter, cystic pouch within the bladder, bilateral hydronephrosis with double ureters, bladder dilated, muscular layers hypertrophied.

Two months before, the patient had a severe attack of hives which disappeared to recur at short intervals. At times her temperature was as high as 104 or 106 F. For four weeks she had complained of severe abdominal pain. Physical examination revealed practically nothing. The lymph nodes were almost universally enlarged.

The urine showed the presence of albumin with numerous pus cells, some of which occurred in clumps. The elevation of temperature continued during subsequent visits. She was placed on alkaline treatment and was later given methenamine and phenyl salicylate. December 15, she had shown no improvement and was sent to the hospital for further investigation. She was placed under the care of Dr. Louis E. Schmidt for urologic examination.

Cystoscopy showed a normal bladder, but large ureteral orifices. An opaque solution, sodium iodide, was introduced into the ureters through catheters, and a roentgen-ray plate was made. The right kidney was enlarged and the pelvis was dilated.

The examination revealed that the patient had dilated ureters on both sides, with an enlarged kidney and pyonephrosis on the right side. It may be assumed that the ureteral dilatation was congenital in origin.

CASE 6.—C. B., a girl, aged 8 years, was admitted to the hospital, Jan. 15, 1926. The child had been ill for the last two years. The condition was supposed to have followed an attack of pleurisy. She was confined to bed for two months with this illness; during this time the urine was examined and was found to contain pus. The pyuria persisted. She had elevation of temperature most of the time, varying from 100 to 102 F. Pus was constantly present in the urine and the child arose twice every night to urinate. The physical examination was negative.

The examination of the abdomen failed to reveal any mass or rigidity. There was no tenderness over the kidneys, the kidneys were not palpable, and there was no tenderness along the course of the ureters.

A roentgenogram of the urinary tract after instillation of an opaque solution by Dr. Harry Culver showed areas of dilatation and constriction in the left ureter, with a stricture at the inferior ureteral opening, probably the result of an inflammatory process. The left kidney was unusually small and appeared to have atrophied. The right ureter and kidney seemed normal. It may be assumed that the pyuria in this case was the result of obstruction in the left ureter. The alternating dilatation and narrowing of the ureter and atrophic kidney were probably due to some acquired inflammatory process.

Eugene Bostroem [9] describes a tumor in a girl, aged 23 weeks. Sixteen weeks before death, it was noted that the abdomen was distended and a three lobe tumor could be palpated. At times considerable quantities of urine were passed, but during the intervals very little urine was eliminated. In this case there is no record of pyuria. Neither is there any record of microscopic urinary examination. The specimen showed congenital obstruction of the right ureter, with the formation of a large cystic pouch within the bladder at the ureteral orifice. The opening of the urethra was displaced by this pouch. There was a bilateral hydronephrosis with double ureters, the bladder was markedly dilated and the muscular layers were hypertrophied.

This case is of anatomic interest, though the clinical observations are not sufficient to enable one to interpret it in terms of symptoms and urinary observations.

CONCLUSIONS

1. Children suffering from acquired or congenital malformations of the urinary tract present a variety of symptoms, such as abdominal pain, fever, chills, emaciation and various disorders of micturition—

9. Bostroem, E.: Beitr. z. Path. Anat., Friebürg and Tübingen, 1884.

dysuria, frequency, polyuria and sometimes anuria. Pyuria is almost a constant symptom.

2. Congenital or acquired malformations of the urinary tract causing dilatation lead to urinary stasis. When stasis occurs, infection is likely to ensue.

3. The pyuria which results runs a protracted course, associated with fever and emaciation, and progressive degeneration of the urinary organs.

4. Every child with protracted pyuria which fails to respond to the usual methods of treatment within a reasonable time, should be subjected to cystoscopic and roentgen-ray examination by a urologist.

THE BASAL METABOLISM AND THE SPECIFIC DYNAMIC ACTION OF FOODS IN CHILDREN IN VARIOUS CONDITIONS OF NUTRITION*

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In connection with a nutritional study of children at the California State Schools for Deaf and Blind, basal metabolism determinations were made on sixteen boys and girls in varying conditions of under and over nutrition. This was done in an attempt to discover any correlation between variations from the average metabolic rate and condition of nutrition. Determinations were made once on four of the subjects, twice at intervals of a month or more on five subjects, and three times at similar intervals on seven subjects. Changes in body weight and stature were taking place in most of these children during this period.

In addition, a comparison of the rise in metabolism following a meal chiefly of protein and one chiefly of carbohydrate was made on four boys in various conditions of nutrition. This was done to see whether leanness and fatness may not be connected with some peculiarity in response of the individual to the specific dynamic action of foods. The latter idea has been tested by a number of investigators, among them Plaut,[1] Liebesny,[2] Wang and Strouse,[3] and Gibbons.[4] All of these observers agree that in obese subjects a lower specific dynamic action is found than in normal or in thin persons. Wang and Strouse attempted to differentiate protein, fat, and carbohydrate meals in this regard. Very few children were included in any of these studies, and in no case were the determinations confined to children alone.

The extensive investigations of Benedict and Talbot [5] have established average figures for the basal metabolism of children up to 12 years of age, which are consistent enough to be used confidently in any compari-

* Received for publication, June 14, 1926.

* From the Laboratory of Household Science, the University of California.

1. Plaut, R.: Gaswechseluntersuchungen bei Fettsucht und Hypophysiserkrankungen, Deutsche Arch. f. klin. Med. **139**:285, 1922.

2. Liebesny, P.: Die Zpezifisch- Dynamische Eiweisswirkung, Biochem. Ztschr. **144**:308, 1924.

3. Wang, C. C., and Strouse, S.: Studies on the Metabolism of Obesity. III. The Specific Dynamic Action of Food, Arch. Int. Med. **34**:1, 1924.

4. Gibbons, R.: On the Specific Dynamic Action of Proteins in Thin and Fat Individuals (Dogs), Am. J. Physiol. **70**:26, 1924.

5. Benedict, F. G., and Talbot, F. B.: Metabolism and Growth from Birth to Puberty, Carnegie Inst. Washington, Pub. 302, 1921.

son of the kind here described. The relations of height to weight, weight to age and height to age were used to establish the normality of the children studied. The relation of height to weight has been emphasized by Benedict and Talbot as most valuable in this regard. Basal metabolism of children referred to weight alone, however, when charted gives points widely scattering, but referred to body surface gives a more concentrated field, and may be considered distinctly helpful in indicating whether a given case shows marked deviation from the general trend which is being tentatively established.

Some of the underweight children observed by Benedict and Talbot showed a higher metabolic rate than did the well-nourished infants. Murlin and Hoobler [6] found the same relation. Blunt and co-workers,[7] who made observations on twenty-eight children, of whom twenty-three were from 9 to 27 per cent underweight, found that ten of the underweight children had basal metabolisms 20 per cent, or more, higher than Benedict and Talbot's norms, while seventeen were 11 per cent, or more, higher. No close relation between degree of undernutrition and excess metabolism was observed.

Of the sixteen children examined in the present study, seven were blind and nine deaf. Their condition of nutrition as measured by the Baldwin and Wood [8] scale varied from 20.9 per cent underweight to 42.4 per cent overweight. Four were 7 per cent, or more, underweight, six were 7 per cent, or more, overweight, and six were within 4 per cent, or less, of the normal figures for height and age. The medical report on these children, as noted by Dr. Rexford Hoobler just previous to the beginning of the study, is given in table 1. Wassermann tests on all the subjects were negative, and, as may be noted in the table, except for tonsils, teeth and adenoids, and the sight or hearing defect, the examination revealed nothing abnormal except in subjects 1, 7 and 12. Subject 12, an overweight boy, was judged to be a hypothyroid case and subjects 1 and 7, underweight girls, were thought to have other glandular disturbances, and in the case of number 7 a congenital heart defect.

The plan of the study involved periodic examinations of these and a number of other children for height, weight, chest circumference, vital capacity, alveolar carbon dioxide content, hemoglobin and basal metabolism, in an attempt to correlate these measurements with the effect of certain supplemental foods given to different groups of the subjects. All

6. Murlin, J. R., and Hoobler, B. R.: The Energy Metabolism of Ten Hospital Children Between the Ages of Two Months and One Year, Am. J. Dis. Child. **9**:81 (Feb.) 1915.

7. Blunt, K.; Nelson, A., and Oleson, H. C.: The Basal Metabolism of Underweight Children, J. Biol. Chem. **49**:247, 1921.

8. Baldwin, B. T., and Wood, T. D.: Mother and Child, 1923, July, suppl. American Child Health Association, Washington, D. C.

the children were given the regular meals served in the dining room of the school, and nothing additional except for the supplemental lunch. Only the basal metabolism measurements are reported in this paper.

METHODS

A Benedict portable respiration apparatus of Sanborn manufacture was used for all determinations. The children were brought to the

TABLE 1.—*Data on Physical Condition of Children*

No.	Sex*	Age, Yr.	Average Height In.	Average Height Cm.	Average Weight Lb.	Average Weight Kg.	Per Cent Under or Over Weight: Baldwin & Wood Standard	Per Cent Under or Over Weight: Pelidisi†	Surface Area A=K$\sqrt[3]{W^2}$‡ Sq. M.	Defect	Medical Report
1	♀	9	42.0	106.7	36.2	16.4	— 7.0	100.2	0.69	Blind	Glandular disturbances, toxic adenoids, carious teeth, cryptic tonsils
2	♀	7	44.4	111.9	43.9	20.0	+ 7.1	98.0	0.79	Blind	Carious teeth, cryptic tonsils, enlarged adenoids
3	♂	7	44.5	113.5	49.7	22.4	+10.4	104.0	0.80	Deaf	Cryptic tonsils, otherwise normal
4	♂	14	59.5	151.1	94.2	42.0	+ 2.4	101.7	1.30	Deaf	Underweight
5	♀	10	52.0	132.0	58.0	26.4	— 9.4	95.7	0.98	Deaf	Underweight, anemic
6	♀	12	50.5	128.2	57.3	26.0	— 9.1	98.7	0.97	Deaf	Underweight but otherwise normal
7	♀	7	39.5	100.3	28.5	12.9	—20.9	89.4	0.59	Deaf	Underweight, congenital heart defect, probable pituitary abnormality
8	♀	10	49.8	126.5	56.2	25.7	— 4.0	96.0	0.96	Deaf	Diffuse adenoids, underweight, anemic
9	♀	10	48.4	122.9	56.2	25.2	+ 3.6	99.5	0.95	Deaf	Carious teeth, otherwise good condition
10	♀	11	49.6	126.1	62.2	28.0	+ 4.9	100.1	1.02	Deaf	Cryptic tonsils, carious teeth, well nourished
11	♀	10	49.4	125.4	63.5	28.7	+11.1	98.6	1.04	Deaf	Enlarged adenoids, good general condition
12	♂	10	51.5	130.8	89.0	40.5	+42.4	107.8	1.35	Blind	Hypothyroidism, overweight
13	♂	10	53.0	134.6	84.0	38.2	+25.3	104.0	1.30	Blind	Adenoids, infected tonsils, good condition of nutrition
14	♂	10	56.0	142.2	78.0	34.5	+ 1.3	98.6	1.22	Blind	Good condition
15	♂	10	53.5	135.9	66.0	30.1	— 3.7	96.0	1.11	Partly blind	Carious teeth, anemic, underweight
16	♂	11	53.5	135.9	77.0	35.0	+11.2	102.7	1.23	Blind	Very bad tonsils, otherwise normal

* In this column, and in table 2, ♀ indicates female; ♂, male.
† Pirquet, 1922.
‡ Benedict and Talbot, p. 62.

metabolism room before breakfast, put to bed and kept there as completely relaxed as possible for thirty to forty minutes before the first test was made. Temperature, pulse and respiration were noted during the rest period as well as during the test. Preliminary determinations were made in all cases to accustom the children to the test before the actual records were made. When a child appeared at all restless or nervous, the test was discarded. Ten minute test periods were used, with five minute readings recorded as well. Duplicate tests, which checked within 3 per cent, were made in all cases. In some, it was necessary to run a third determination in order to secure this agreement. Rest periods of at least twenty minutes were allowed between tests.

The apparatus was tested for leaks before every observation, and for unabsorbed carbon dioxide after every observation. With the help of Carpenter's [9] tables, total calories per twenty-four hours were computed from the average oxygen consumption per minute observed. These results are given in table 2, along with the standards calculated by Benedict and Talbot [5] from body weight, and standards read from their curves for basal metabolism referred to body surface.

TABLE 2.—*Basal Metabolism of Sixteen Children*

				Weight		Basal Metabolism in Calories per 24 Hours					Per Cent Variations from Basal Metabolism Standards			
						Observed		Predicted			By Weight			
No.	Sex	Age, Years	Date, 1925	Per Cent Difference from Normal	Average	Readings	Average	From Weight	From Surface	From Height (Girls)	Difference	Average	By Surface	By Height (Girls)
1	♀	9	3/31	— 7.0	— 7.0	791	791	722	720	737	+ 9.5	+ 9.5	+ 9.9	+ 7.3
2	♀	7	3/ 6	+ 5.5	+ 7.1	793	801	801	790	787	+ 0.1	— 0.3	+ 1.4	+ 1.8
			3/27	+ 8.5		799		812			— 1.5			
			4/22	+ 7.3		812		805			+ 0.8			
3	♂	7	3/30	+11.1	+10.4	1,045	1,020	931	930	...	+12.2	+ 9.8	+ 9.6	.....
			4/ 1	+11.1		1,054		931			+13.2			
			5/12	+ 8.9		960		924			+ 3.9			
4	♂	14	4/ 1	+ 0.8	+ 2.0	1,294	1,290	1,306	1,345	...	— 0.9	— 1.5	— 4.0	.....
			4/27	+ 3.8		1,311		1,318			— 0.5			
			5/19	+ 2.7		1,266		1,308			— 3.2			
5	♀	10	3/ 3	— 9.4	— 9.4	1,116	1,127	960	950	985	+16.2	+17.4	+18.6	+14.4
			4/20	— 9.4		1,139		960			+18.6			
6	♀	12	3/ 3	—10.3	— 9.1	966	950	950	945	945	+ 1.6	— 0.2	+ 0.4	+ 0.5
			4/20	— 9.9		930		952			— 2.3			
			5/15	— 9.1		956		955			± 0.0			
7	♀	7	3/31	—20.9	—20.9	640	640	631	620	678	+ 2.8	+ 3.8	+ 4.6	— 4.2
8	♀	10	3/ 2	— 2.6	— 4.0	1,037	974	938	940	930	+10.5	+ 3.6	+ 3.7	+ 4.7
			3/19	— 5.1		937		938			± 0.0			
			4/21	— 4.3		948		944			+ 0.4			
9	♀	10	3/ 4	+ 5.6	+ 3.6	844	854	938	920	893	—10.0	— 9.2	— 7.1	— 4.3
			4/22	+ 1.8		847		932			— 9.1			
			5/22	+ 3.6		870		950			— 8.4			
10	♀	11	2/28	+ 4.2	+ 4.9	1,034	1,026	922	980	926	+ 4.2	+ 2.2	+ 4.7	+10.8
			3/17	+ 6.4		1,019		1,005			+ 1.4			
			4/21	+ 4.1		1,027		1,018			+ 0.9			
11	♀	10	3/27	+10.7	+11.1	1,022	1,000	1,002	1,000	919	+ 1.9	— 1.7	± 0.0	+ 8.8
			5/14	+11.6		977		1,032			— 5.3			
12	♂	10	5/ 7	+42.4	+42.4	1,134	1,124	1,320	1,310	...	—14.1	—14.9	—14.2	.....
			5/11			1,113					—15.7			
13	♂	10	5/21	+25.3	+25.3	1,065	1,065	1,275	1,270	...	—16.4	—16.4	—16.1	.....
14	♂	10	5/21	+ 1.3	+ 1.3	1,216	1,216	1,230	1,200	...	— 1.1	— 1.1	+ 1.3	.....
15	♂	10	5/ 7	— 3.7	— 3.7	1,217	1,206	1,115	1,105	...	+ 9.2	+ 8.1	+ 9.1	.....
			5/11	— 3.7		1,195					+ 7.1			
16	♂	11	1/28	+ 9.4	+11.2	1,071	1,087	1,200	1,210	...	—10.7	— 9.1	—10.1	.....
			3/11	+12.4		1,104		1,200			— 9.5			

Weights were taken with shoes and coats or sweaters removed, and these weights were corrected as suggested by Baldwin and Wood to corresponding nude weights. Standing heights were taken without shoes and in the manner recommended by the United States Public Health Service.

9. Carpenter, T. M.: Tables, Factors, and Formulas for Computing Respiratory Exchange and Biological Transformations of Energy, Carnegie Inst. Washington, Pub. 303, 1921.

The surface area of the subjects was calculated from the formula $A = K \sqrt[3]{w^2}$, using the values for K proposed by Benedict and Talbot.[5] These figures are given in table 1, and in table 2 are shown the theoretic basal metabolisms corresponding with these surface areas as read from the smoothed curve of Benedict and Talbot [5] (figs. 38 and 39). In addition, the new figures for probable metabolism of girls as predicted from height by Benedict [10] are also given for the nine girls.

The percentage divergences of all observed metabolisms from these three standards, predicted from weight, surface and height respectively, are compared in table 3 with the condition of nutrition of the subjects.

TABLE 3.—*Comparison of Condition of Nutrition and Basal Metabolism*

Condition of Nutrition	Number of Children: Total	Number of Children: Possibly Abnormal	Number of Determinations	Per Cent Difference from Standard Basal Metabolisms: Weight	Surface (Benedict and Talbot)	Height (For Girls)
Normal						
Range.....	−4.9 to +4.0	6	0	14	− 9.2 to +8.1	− 7.1 to +9.1	− 4.3 to +10.8
Average...	−0.5				−0.7	+1.3	+3.7
Underweight						
Range.....	−7.0 to −20.9	4	2	7	− 0.2 to +17.4	+ 0.4 to +18.6	− 4.2 to +14.4
Average...	−11.6				+7.6	+8.9	+4.5
Overweight						
Range.....	+7.1 to +42.4	6	2	13	−16.3 to +9.8	−16.1 to +9.6	+1.8 and +8.8
Average...	+17.9				−5.4	−5.0	+5.3

RESULTS AND COMMENT

In the ten cases in which two or three determinations were made a month or more apart, a tendency toward somewhat lower metabolism in the late spring is observable. There is only one exception to this, that of number 4, a boy approaching puberty, in whom considerable fluctuations of weight were taking place. Whether this seasonal factor is one of general import cannot be stated until more data are available. The same seasonal effect was observable in certain other measurements, particularly that of plasma bicarbonate [11] made on these and on other children in the same institution and at the same time.

Of the four girls, numbers 1, 5, 6 and 7, who were 7 per cent, or more, underweight, two, numbers 1 and 5, had 7.3 and 14.4 per cent excess metabolism as measured by the height standard of Benedict. The other two, numbers 6 and 7, showed practically normal metabolisms. Of the six children, numbers 2, 3, 11, 12, 13 and 16, who were 7 per cent, or more, overweight, four, numbers 11, 12, 13 and 16, had

10. Benedict, F. G.: Physical Factors in Predicting the Basal Metabolism of Girls, Proc. Am. Phil. Soc. **63**:25, 1924.

11. Morgan, A. F., and Hatfield, G. D.: The Effect of Various Supplementary Lunches upon the Plasma Carbon Dioxide Capacity of Children, Am. J. Dis. Child., to be published.

metabolisms less than that predicted from the weight standards of Benedict and Talbot. Number 12 was a hypothyroid case, but the others, with the possible exception of number 3, were apparently normal. Number 3, a boy of 7, who, according to the Baldwin-Wood table, was 10 per cent overweight for his height, was nevertheless classed by the medical examiner as underweight, and by repeated plasma bicarbonate tests as reported in another place was shown to be distinctly acidotic. A certain amount of hyperpnea was evident in this subject during all tests. His metabolism was 9.8 and 9.6 per cent above the normal, according to weight and surface standards. Number 2, although 7 per cent overweight, appeared to be a normal well-nourished child, and gave a metabolism close to the standards.

The other six children observed, numbers 4, 8, 9, 10, 14 and 15, who were close to the average weight for height, gave fairly good agreement with the standard.

It is interesting to note that the boy with supposed hyperthyroidism, number 12, showed no greater lowering of the metabolism than did the obviously normal, but similarly overweight boy, number 13. Likewise the extremely underweight girl, number 7, who was thought to have a pituitary abnormality, showed a practically normal metabolism. On the whole, our results offer some slight confirmation of the conclusions of Benedict and Talbot, and of Blunt and co-workers, that undernourished children are apt to show higher metabolism than normal children. On the other hand, our figures for overweight children rather convincingly support the supposition that such children have lower than normal basal metabolic rates. For our normal weight children the rates observed were well within the 10 per cent variation from standard.

When the average condition of nutrition is compared with the deviation of basal metabolism from a standard based on the Benedict and Talbot surface formula, much better agreement with the current opinions as to these relations is found than when weight standards are used.

The height standards for girls proposed by Benedict offer no advantage of regularity over the surface figures, and in some cases, such as numbers 7, 10 and 11, indicate considerably greater divergence from the observed values than do the weight and surface standards. Subjects 10 and 11 were undoubtedly normal girls, but subject 7 may have had some glandular irregularity.

THE SPECIFIC DYNAMIC ACTION OF FOODS IN OVERWEIGHT AND IN NORMAL WEIGHT CHILDREN

Four 10 year old boys, numbers 12, 13, 14 and 15, were selected as subjects for the experiment dealing with the specific dynamic action of foods. According to the Baldwin and Wood standards, numbers 12 and 13 were 42.4 and 25.3 per cent, respectively, overweight for their

height and age, and numbers 14 and 15 were of normal weight, being respectively 1.3 per cent over and 3.7 per cent under the standard. There was some suspicion that number 12 was hypothyroidic, but his basal metabolism did not markedly confirm this diagnosis. The other three boys were entirely normal active children.

Basal metabolism was determined as previously described early in the morning of the test. The carefully weighed test meals were then given, and after two and one-half hours metabolism determinations made again. Half-hour rest periods preceded the second determinations as well as the first.

RESULTS

As shown in table 4, two types of test meal were used, namely, one of predominantly carbohydrate content, the other chiefly protein in character. However, only 68.5 per cent of the calories in the first meal were

TABLE 4.—*Composition of Test Meals*

Food	Weight, Gm.	Protein, Calories	Fat, Calories	Carbohydrate Calories	Total Calories
Carbohydrate Meal:					
Prunes	140	10.1	0.0	349.3	359.4
Cereal	150	25.6	10.7	113.2	149.6
Milk	50	8.6	10.5	15.3	34.7
Sugar	25	0.0	0.0	100.0	100.0
Butter	23	2.0	174.8	0.0	176.8
Bread	104	41.8	50.8	168.4	265.2
Total meal, calories		88.4	255.8	741.3	1,085.7
Total percentage		8.1	23.5	68.5	
Protein Meal:					
Milk	350	65.1	78.5	98.5	242.1
Ham	50	53.2	125.3	0.0	178.5
Eggs	150	180.5	141.5	0.0	322.0
Bread	20	8.2	1.2	43.3	52.7
Butter	10	0.9	76.0	0.0	76.9
Total meal, calories		307.9	422.5	141.8	872.2
Total percentage		35.6	48.4	16.0	

furnished by carbohydrate, 23.5 per cent by fat and 8.1 per cent by protein. In the protein meal only 35.6 per cent of calories came from protein, 48.4 per cent from fat and 16 per cent from carbohydrate. It is obvious that widely divergent figures for the stimulating effects of these two meals could hardly be expected. There was, however, more than twice the increase in metabolism resulting from the protein meal in both subjects as compared with that obtained from the carbohydrate meal. These figures are shown in table 5. Only small differences are observable between the responses of the overweight boys, numbers 12 and 13, and the normal boys, numbers 14 and 15, to the stimulating effects of the meals eaten. The actual amount of increase is comparable with the figures obtained by Wang and Strouse [3] on their one child subject, E. M., aged 12½ years, 94 per cent overweight, who showed 9.8 per cent increase in metabolism two hours after a protein meal. Our data are too few to justify conclusions, but would seem to point toward less obvious differences in response to food stimulation of

metabolism in underweight and overweight children than in adults of similar weight type.

SUMMARY

1. The basal metabolism of sixteen children, from 7 to 14 years of age, in varying states of nutrition was determined. Ten of the subjects were tested two or three times at intervals of a month or more. There is an apparent lowering in metabolism in the spring as compared with the winter measurements. The total heat production per twenty-four hours of these children is compared with the metabolism by weight and surface standards proposed by Benedict and Talbot, and with the metabolism by height standards for girls proposed by Benedict.

2. The six children classed as of *normal* weight by the Baldwin and Wood standards, showed metabolisms falling within a ± 10 per cent

TABLE 5.—*Specific Dynamic Effect of Protein and Carbohydrate Meals on Normal and Overweight Boys*

Number	Age, Years	Weight, Kg.	Per Cent Under or Overweight	Basal Metabolism: Date, 1925	Basal Metabolism: Calories per 24 Hours	Per Cent Under or Over Normal Basal Metabolism From Weight	Metabolism After Meal, Calories per 24 Hours	Per Cent Above Basal
							Carbohydrate meal	
15	10	30.1	— 3.7	5/ 5	1/217	+ 9.2	1,292	6.1
12	10	40.4	+42.4	5/ 5	1,134	—14.1	1,188	4.7
							Protein meal	
15	10	30.1	— 3.9	5/11	1,195	+ 7.1	1,355	13.4
14	10	35.4	+ 1.3	5/11	1,216	— 1.1	1,348	10.8
12	10	40.4	+42.4	5/11	1,113	—15.7	1,244	11.7
13	10	38.1	+25.3	5/11	1,066	—16.4	1,163	9.2

deviation from standards by weight and by surface area. Two of the four girls classed as *underweight* showed nearly normal metabolisms by all standards. The other two had high metabolic rates. Of the six children classed as *overweight* for height and age, three showed abnormally low metabolisms, two had normal metabolisms, and one, possibly abnormal, 9 per cent above the standards. Thus ten out of the sixteen children exhibited basal metabolisms within 10 per cent of the weight and surface area standards of Benedict and Talbot.

3. Three of the nine girls examined showed wider divergences from the height standard of Benedict than from the older weight and surface area standards.

4. Four boys, aged 10 years, two distinctly overweight and two of normal weight, were tested for increase in metabolism following carbohydrate and protein meals. More than twice as much increase was noted after the protein meal as compared with that after the carbohydrate meal, but only small and irregular differences between the increases shown by the overweight and normal boys were obtained. More data are needed before conclusions concerning such differences found in adults can be confidently applied to children.

FRUIT AND VEGETABLE JUICES USED IN INFANT FEEDING

A COMPARISON OF THEIR GROWTH PROMOTING QUALITIES *

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AND
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A series of experiments were planned to show the comparative growth promoting qualities of some of the fruit and vegetable juices recommended for infant and child feeding.

Since milk has been found by experiment to vary in vitamin content according to the food of the cow and the treatment the milk has undergone previous to feeding,[1] it has been customary to give orange juice to supplement the milk diet.[2] Orange juice stimulates growth, prevents scurvy, furnishes a considerable quantity of minerals, and has laxative qualities. Oranges, however, are difficult to obtain in some rural communities, and at some seasons of the year they are too expensive to be available to everyone. Many authors have pointed out recently that the same purposes would be served by other and cheaper fruit and vegetable juices. Those juices most commonly recommended are tomato, cabbage, spinach and carrot. All these compare favorably with orange in mineral content, and with the possible exception of carrot, they appear to be comparable in their ability to prevent scurvy.[3] These have all been

* Received for publication, Aug. 5, 1926.

1. Funk, Casimir: The Vitamins, Baltimore, Williams & Wilkins Company, 1922, pp. 256, 264, 282.

2. Carter, H. S.; Howe, P. E., and Mason, H. H.: Nutritional and Clinical Dietetics, Philadelphia, Lea & Febiger, 1921, p. 287. Davey, Alice J.: Determination of the Minimum Doses of Some Fresh Citrus Fruit Juices Which Will Protect a Guinea-Pig from Scurvy, Biochem. J. **15**:83, 1921. Davies, Margaret, and others: Observations on Vitamin Content of Foods, J. Home Economics **12**:209, 1920. Drummond, J. C.: Researches upon the Fat-Soluble Accessory Substance. 1. Observations upon Its Nature and Properties, Biochem. J. **13**:81, 1919.

3. The following amounts of fruit and vegetable juices have been found necessary to prevent scurvy in a guinea-pig: 1.5 cc. fresh orange juice (Davey, A. J.: Biochem. J. **15**:83, 1921); 2 to 5 cc. canned tomato juice (Delf, E. M.: Biochem. J. **18**:674, 1924; Sherman, H. C.; La Mer, V. K., and Campbell, H. L.: Am. Chem. Soc. J. **44**:170, 1922); 1 to 1.5 Gm. raw cabbage (Eddy, W. H.; Shelow, E., and Pease, R. A.: Proc. Soc. Exper. Biol. & Med. **19**:187, 1921; Campbell, M. E., and Chick, H.: Lancet **2**:321, 1919). 1 Gm. raw spinach or 4 oz. canned spinach (Eddy, W. H.; Kohman, E. F., and Carlsson, V.: Indust. & Engin. Chem. **16**:53, 1924; ibid. **17**:69, 1925).

The report of the Medical Research Council (ed. 2, London, 1924), gives the following amounts: Raw fresh cabbage, 1 Gm.; raw young carrots, 10 Gm.; orange juice, 1.5 cc.; raw tomatoes, 1.5 cc.; canned tomatoes, 3 cc.

tested for their vitamin content, but as yet no direct comparisons have been made.

This investigation was undertaken, therefore, to see if these vegetables, which are easier and cheaper to procure, are equivalent to oranges in growth promoting qualities when fed in equal amounts. If so, the problem of infant feeding among the poor and isolated families is very much simplified.

EXPERIMENTAL

In these experiments, a basal diet was selected which was the same for all of the animals, and the fruit or vegetable to be tested was fed separately. The basal diet supplied vitamin A but no vitamin B, and since rats are but slightly susceptible to scurvy, vitamin C was not taken into consideration. The diet used was similar to the diets used by other investigators.[4] It consisted of 18 per cent casein, 3 per cent salt mixture (Gulick's formula), 3 per cent agar, 17 per cent crisco, 3 per cent butter fat, 38 per cent cornstarch and 18 per cent cane sugar. This diet did not support growth.

The usual care was taken to secure quantitative feeding and to prevent the consumption of feces. Young albino rats (weighing from 40 to 80 Gm.) were used for the experiment. They were cared for in accordance with the methods recommended by Ferry.[5] Square cages of wire mesh were used, which allowed the fecal material to fall through onto folded paper in the tray, thus preventing its consumption, and also allowing for the recovery of any spilled food. Food for one week for each rat was weighed, and placed in a glass bottle with the number of the animal written on the bottle. Another bottle was placed by each of these to collect the spilled food. At the end of each week all food not eaten was weighed. The animals were weighed every third day, and accurate records of their weights and food consumption were kept. The fruit or vegetable juice was fed separately from the basal ration.

The method of preparing the fruit juices differed according to the food. The oranges were a well known California brand. The same brand was used throughout the experiment so as to insure as much uniformity as possible. The juice was squeezed out and strained.

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4. Hawk, Philip B.: Physiological Chemistry, Philadelphia, P. Blakiston's Son & Company, 1923, p. 592. Miller, Carey D.: Vitamins in Pineapple, J. Home Economics **16**:18, 74, 1924. Osborne, T. B., and Mendel, L. B.: Quantitative Aspects of the Rôle of Vitamin B in Nutrition, J. Biol. Chem. **54**:739, 1922; Pub. 156, Carnegie Inst. Bull. **1, 2,** 1911. Osborne, T. B.; Wakeman, A. J., and Ferry, E. L.: Preparation of Protein Free from Water-Soluble Vitamin, J. Biol. Chem. **39**:35, 1919. Steenbock, H.; Sell, M. T., and Jones, J. H.: Storage of Vitamin B by the Rat, J. Biol. Chem. **55**:411, 1923.

5. Ferry, E. L.: Osborne and Mendel's Technique on the Feeding of Rats, J. Lab. & Clin. Med. **5**:735, 1920..

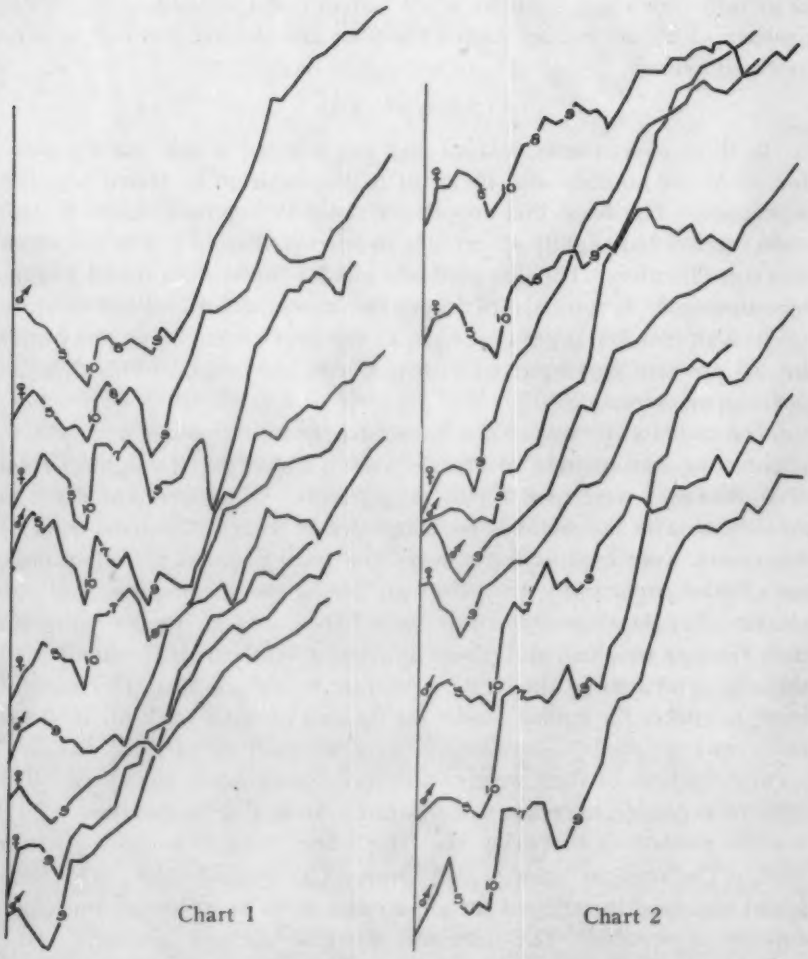

Chart 1.—Growth curve of rats fed orange juice. In each chart, the figures show the amount in cubic centimeters of juice fed.

Chart 2.—Growth curves of rats fed tomato juice.

The same medium grade brand of tomatoes was used throughout the experiment. They also were strained, and only the juice was given.

The cabbage was chopped; then the juice was expressed and strained.

The spinach was weighed, washed, and boiled for 20 minutes without adding water, except what adhered to the leaves from washing. The juice was expressed, and from one pound of spinach 367 cc. of juice was obtained. Hence, 9 cc. of juice was equivalent to 11.1 Gm. of raw spinach.

Chart 3.—Growth curves of rats fed spinach juice.

The carrots were weighed, cut in small pieces, covered with water, and boiled until tender. The juice was expressed from the carrots and added to the cooking liquid. From one pound of freshly cut carrots 181 cc. of juice was obtained. Therefore, 9 cc. of juice was equivalent to 22.5 Gm. of carrot. The spinach and carrot juices were prepared once a week, placed in sealed containers, and put in the icebox until used. Nine rats received orange juice; nine, tomato juice; five, spinach juice; five, carrot juice, and five cabbage juice.

A preliminary series of experiments with amounts of orange and tomato juices varying from 5 to 10 cc. showed that 9 cc. was necessary

to support normal growth, and no distinction could be drawn between the orange and tomato.

Therefore, 9 cc. of each of the juices used was fed daily, and the growth obtained with the carrot and spinach juices compared favorably with that obtained with tomato and orange. Cabbage juice, however, did not support growth, and three of the five rats died before the end of the experiment. It occurred to us that probably fresh cabbage would be more effective than that stored throughout the winter, and when the fresh cabbage came into the market, four rats were each given 9 cc. of it. They grew well during the 50 days that they received the diet.

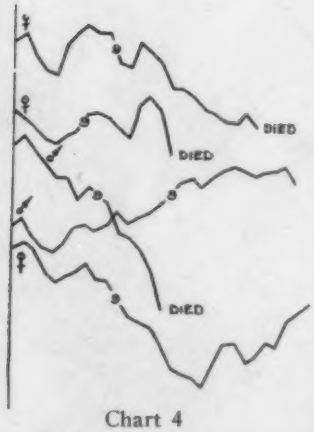

Chart 4

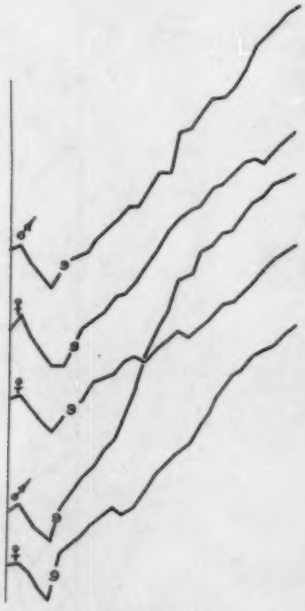

Chart 5

Chart 4.—Growth curves of rats fed cabbage juice (winter).
Chart 5.—Growth curves of rats fed carrot juice.

SUMMARY

A series of experiments were carried out to determine the comparative growth promoting qualities of several fruit and vegetable juices which have been recommended for child and infant feeding. These indicate that physicians and research workers are justified in substituting carrot, spinach, or tomato juice for the conventional orange juice. Winter cabbage does not seem to be as satisfactory for this purpose as the other vegetables studied.

These experiments did not show whether carrot or spinach juice is superior to orange or tomato juice, as no attempt was made to determine

the minimum amounts of these juices which would support normal growth and nutrition in the rat.

In these experiments, we have observed that the maturity of the vegetable and the length of time it has been stored appear to affect its growth promoting qualities. No special variety of carrots was fed, and occasionally large, mature, or carrots long in storage were received. It was observed that these did not produce as much growth as the small young carrots.

The cabbage which was used in the first part of the experiment was winter cabbage, and contained very little juice. Fresh spring cabbage was used as soon as it could be obtained. This yielded more juice and supported better growth than the winter cabbage.

CONCLUSION

The minimum amount of orange or tomato juice which would support normal growth in the rat was found to be 9 cc. The same quantity of spinach or carrot juice gave equally good results. Nine cubic centimeters of winter cabbage did not support growth, but during a short experiment, in which fresh summer cabbage was used, it was seen to be comparable in growth promoting power to the other juices used.

URIC ACID IN THE URINE OF INFANTS

II. DAY AND NIGHT EXCRETION *

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In a previous paper,[1] I reported the results of a study of endogenous uric acid in the urine of infants. It was found that the day by day elimination of uric acid was fairly constant for each person, the total output ranging from 15 to 25 mg. per kilogram of body weight in twenty-four hours. The present work deals with the diurnal and nocturnal excretion of uric acid in five healthy infants.

The difficulties which are encountered in making quantitative twenty-four hour collections of infants' urine for metabolic studies are still greater when collections are made for shorter, successive periods. Owing to the intensive water exchange, a healthy infant normally voids a relatively large amount of urine, and empties his bladder at much more frequent intervals than an adult, due to his lack of control of the muscles regulating micturition. However, the evacuation of the bladder does not occur at regular intervals and it is, therefore, difficult to adapt them to short metabolic periods. There is perhaps no way of collecting infants' urine quantitatively for shorter periods unless catheterizations are done at the beginning and at the end of each period. We did not feel justified in adopting this procedure, so our results are based on the analysis of three hour urine samples, voided spontaneously by the infants studied. The objection which can be raised to this method is: Does the total amount of urine and uric acid voided from the bladder during each three hour period correspond to the amount actually excreted by the kidneys? We hoped that the selection of subjects who were comparable in age and habits and who lent themselves best to accurate collections would minimize this objection. It was soon found that younger infants showed the best cooperation in these short period collections. The older infants were not well adapted to our work because of the long intervals between voidings. The analyses of their extremely variable samples gave such discrepant figures that no conclusions could be drawn concerning the regularity of uric acid excretion in short periods.

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* Received for publication, April 22, 1926.

* From the infants' wards of the New York Nursery and Child's Hospital, and the Department of Pediatrics, Cornell University Medical College.

1. Rougichitch, O. S.: Uric Acid and Creatinine in the Urine of Infants, Am. J. Dis. Child. **31**:504 (April) 1926.

PROCEDURE

Five healthy infants between the ages of 6 and 12 weeks were studied. They were all male infants, as the method of collecting the urine could not be applied to the female infants. They received the usual routine hospital care. Their diets were dilutions of sweet or acidified cow's milk, and their total caloric intakes were suited to their requirements. The feedings remained unchanged for several days before, during and after the collections.

The routine of the experiments was as follows: The method of collecting urine samples was the same as described in the previous paper. No preperiod was run. The urine was collected in three hour periods during the day and night, starting at 8:00 a. m. The bottle was withdrawn exactly at the end of each period. Following one day of fractional collections, one total twenty-four hour collection was made as a check on the total uric acid excretion. In all cases reported no urine was lost. Urine samples were preserved on ice and by the addition of toluene. Uric acid measurements were made within the first twenty-four hours after collection, using Folin's [2] microchemical colorimetric method. The results are shown graphically in the table and in the chart.

The day and night endogenous uric acid excretion has been extensively studied by many investigators, but almost all of these studies deal with adults. Pfeil,[3] Osterberg and Wolf,[4] Rockwood,[5] and others, found that in adults the uric acid elimination was greater during the day than during the night. According to their observations, the hourly excretion of uric acid reaches the maximum in the forenoon, followed by a progressive decline until early the next morning when an increase to the maximum again occurs. Various explanations for this have been offered, but none seems to be entirely satisfactory. In 1924, Leopold, Bernhard and Jacobi,[6] studying five normal children between the ages of 7 to 12 years, observed that the two hour endogenous uric acid excretion was highest between 10 and 12 a. m., followed by a progressive decrease until the minimum is reached at 6:30 p. m., with a subsequent rise at 8:00 p. m. Their figures for the day and night ratio show that the endogenous uric acid output in their cases was greater during the day than during the night. They did not offer any explanation for that phenomenon. I have found no published data bearing on a study on infants on this subject.

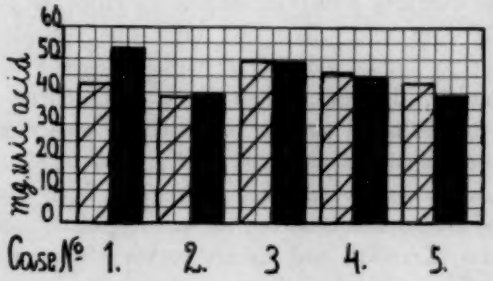
2. Folin, O.: Lab. Man. Biol. Chem., 1923.
3. Pfeil: Ztschr. f. phys. Chem. **40**:1, 1903.
4. Osterberg and Wolf: J. Biol. Chem. **3**:169, 1907.
5. Rockwood, E. W.: Am. J. Physiol. **12**:38, 1904.
6. Leopold, J. S.; Bernhard, A., and Jacobi, H. G.: Studies in the Uric Acid Metabolism of Child., Am. J. Dis. Child. **27**:243 (March) 1924.

Uric Acid Excretion in Five Healthy Young Infants, Comparing the Day and Night Amounts in Three Hour Periods

Case; Age; Weight	Day (1926) and Analysis	Total in 24 Hours	Total Day	Total Night	Three Hourly Periods: 8 a. m. to 11 a. m.	11 a. m. to 2 p. m.	2 to 5 p. m.	5 to 8 p. m.	8 to 11 p. m.	11 p. m. to 2 a. m.	2 to 5 a. m.	5 to 8 a. m.	Feeding
1	3/23-3/24												
6 weeks	Uric acid, mg.	96.3	42.4	53.9	....	15.3	18.7	8.4	12.4	14.6	6.0	20.9	Lactic acid
3.4 Kg	Urine, cc.	218	63	155	....	28	21	14	32	34	13	76	milk mixture
	3/24-3/25												
	Uric acid, mg.	99.4											
	Urine, cc.	146											
2	1/30-1/31												
8 weeks	Uric acid, mg.	77.2	37.8	39.4	7.4	9.7	4.4	16.3	4.9	10.8	13.6	10.1	Whole milk
4.3 Kg.	Urine, cc.	355	159	196	60	30	12	57	0	..	90	78	mixture
	1/31-2/1												
	Uric acid, mg.	80.9											
	Urine, cc.	308											
3	3/21-3/22												
8 weeks	Uric acid, mg.	90.0	49.7	40.3	9.4	9.8	19.8	10.7	14.3	8.5	14.9	11.6	Whole milk
4.3 Kg.	Urine, cc.	459	231	228	56	57	83	45	85	42	51	60	mixture
	3/22-3/23												
	Uric acid, mg.	94.8											
	Urine, cc.	364											
4	1/3-1/4												
10 weeks	Uric acid, mg.	90.8	45.8	45.0	10.7	12.8	9.7	12.6	13.8	5.4	10.0	15.8	Whole milk
5.5 Kg.	Urine, cc.	566	243	323	34	90	48	72	99	22	87	115	mixture
	1/4-1/5												
	Uric acid, mg.	94.6											
	Urine, cc.	360											
5	3/18-3/19												
12 weeks	Uric acid, mg.	81.6	42.8	38.8	12.8	9.4	8.7	11.9	10.5	8.1	9.4	10.8	Whole milk
5.2 Kg.	Urine, cc.	242	82	160	20	22	16	24	37	33	54	36	mixture
	3/19-3/20												
	Uric acid, mg.	86.8											
	Urine, cc.	286											

DAY AND NIGHT URIC ACID RATIO

The most striking feature noted in the figures from the infants in this study is that the ratios between the day and night uric acid excretion are not in accord with those found in studies of adults and children. None of our cases gave evidence of the characteristic morning rise observed in adults and children. The curve of elimination in cases 1, 2 and 3 is rather irregular, the irregularities occurring at different times of the day, while it is notably regular in cases 4 and 5. It is easy to observe that these irregularities occur during the periods of irregular micturitions, as evidenced by the varying amounts of urine voided during corresponding periods. These maximum and minimum figures are secured from samples in adjoining periods, and probably represent overlapping due to the carrying over of unvoided urine into the following period. It is remarkable, however, that the amount of



The day and night ratio of uric acid excretion in five normal infants between the ages of 6 and 12 weeks. Night excretion, black column; day excretion, black and white column.

uric acid excreted is more regular than the volume of urine voided in each period. We feel that it would be unjustifiable to draw any definite conclusions concerning the three hour uric acid excretion because of this demonstrated difficulty in securing uniform collections over short periods of time. However, the day and night uric acid ratio, as seen in our table, is about equal. We wish to emphasize the fact that this ratio covers two longer periods of twelve hours each, thus diminishing the source of possible error due to incomplete evacuation of the bladder.

COMPARISON OF ADULT'S AND INFANT'S URIC ACID EXCRETION

Why are the diurnal and nocturnal endogenous uric acid excretion equal in these cases, and what is an adequate explanation of that fact? Attempts have been made at various times to relate the variations in the hourly uric acid excretion of adults to their daily habits. Since an infant's mode of life is decidedly unlike that of an adult, a consideration of the differences may prove of value in this study.

Burian's [7] contention, that muscular exercise increases the endogenous uric acid excretion only when strenuous work is done, is today generally admitted, while it has been found that there is no increase with ordinary exercise. Campbell and Webster's [8] studies of the day and night endogenous uric acid excretion under "rest routine" and "ordinary routine" showed similar curves in each state. Though the bodily activity of the infants studied was roughly the same during the day and night, this factor does not seem to be sufficient to explain the observations in our cases.

Another factor which has been suggested is the possible interrelation between the uric acid output and the size, composition and spacing of meals. The fact that an infant in the age period which we studied takes food of an unvarying composition and constant caloric value in feedings, quite equally spaced through the twenty-four hours, might be considered as exerting a distinct stabilizing influence on the day and night output of uric acid. We find only one recorded attempt to subject an adult to similar conditions while studying his endogenous uric acid elimination. Leathes,[9] when studying the curve of elimination of uric acid, kept his adult subject on a purine free diet of sufficient caloric value, and distributed the food over six meals, all of which were identically the same and were given at regular intervals. He found that the curve of elimination obtained under these circumstances was still typical of adults. Leonard and his co-workers found the same to be true in their studies, even when their patients were on a starvation diet.

There has been discussion in the literature as to whether there is any close relationship between the volume of urine voided and the amount of uric acid excreted, and probably, according to recent studies, the results are against that view. Neuwirth [10] thinks, however, that this ratio does exist within certain limits. Furthermore, he found in his studies on adults, that, with a constant intake of a fairly liberal quantity of water, the hourly excretion of uric acid is somewhat more regular. As an infant's fluid intake is normally higher and more regular than that of an adult, this may be considered as a possible explanation.

COMMENT

There is not enough evidence that any of the before-mentioned factors, individually or collectively, could satisfactorily explain our observations. The regularity of uric acid day and night excretion in these cases probably could not be related to the regularity of renal

7. Burian, R.: Ztschr. f. phys. Chem. **43**:532, 1904-1905.
8. Campbell, J. A., and Webster, T. A.: Biochem. J. **15**:660, 1922.
9. Leathes, J. B.: J. Physiol. **35**:125, 1906.
10. Neuwirth: J. Biol. Chem. **29**:477 (April) 1917.

activity, for, as observed in adults, the rate of uric acid excretion is lowest during the periods of highest renal activity, as estimated by total nitrogen excretion. We must, therefore, turn to the cellular metabolism, and, with Leathes, look for an explanation in the resulting breaking down of the nucleic compounds and the rate of uric acid formation and destruction within the body. Whatever the explanation may be, our results show a remarkable regularity of the purine metabolism of infants.

SUMMARY

The urinary uric acid three hour diurnal and nocturnal excretion in five healthy infants was studied. Three infants showed considerable irregularity in the uric acid found in the separate samples, probably because of overlapping of periods. Two infants showed a remarkable uniformity in the rate of excretion in successive three hour periods. The total diurnal output of uric acid was strikingly similar to the total nocturnal excretion in all five cases. This is an interesting contrast to the diurnal maximum and nocturnal minimum present in adults.

161 West Sixty-first Street.

THE INCIDENCE OF ENDOCARDITIS IN EARLIEST CHILDHOOD*

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INTRODUCTION

In the study of heart disease in the present, as in the past, one of the most important problems is that of the origin of chronic valvular disease in young people. Especially is this true now, since definite steps have been taken to organize activities directed toward the prevention of heart disease.

In many cases it is, of course, easy to trace the onset of valvular deformity to some clear-cut attack of rheumatic fever or chorea. Often, it is more difficult to prove the occurrence of such infections in young people with chronic endocarditis, especially if the rheumatic fever or chorea have been very mild and the heart has not been carefully watched during and directly after the infection. Sometimes it is impossible to obtain any history of such illness in patients with chronic valvular disease in youth. Because rheumatic fever and chorea are the commonest known causes of such endocarditis, and because physical signs and pathologic changes appear to be much the same in patients with chronic rheumatic valvular disease and in those with valvular disease of unknown origin, it has become the custom today to consider all chronic valvular disease (except that of congenital origin) in young people to be the result of rheumatic infection. This custom has the advantage of calling our attention more forcibly to the importance of the campaign against rheumatic fever and chorea, but as Dr. Henry Jackson of Boston has declared recently, the method is not a very scientific one. We should still speak of endocarditis or valvular disease as of unknown origin in some cases, rather than assure ourselves that the cause in such instances must be rheumatic. Perhaps it is all right to label these conditions as belonging to the "rheumatic type" of heart disease, but by so doing we must not blind ourselves to the need of getting more information than we now possess concerning their origin. Dr. Jackson, who is one of our older and most discerning internists, tells me of his experience of having been taught in youth that valvular disease was always rheumatic; then came the idea that sometimes septic

* Received for publication, May 10, 1926.
* From the Children's Cardiac Clinic, Massachusetts General Hospital.
* Read before the Medical Society of the County of New York, March, 22, 1926.

infection could produce it, and now once more there is the tendency to regard it entirely as rheumatic in origin. In 1912, Still [1] wrote:

> Perhaps it would be rash to say that rheumatism is the only cause of endocarditis in childhood, but my own experience strongly suggests this. Out of 150 cases in which I have investigated this point, 142 showed evidence of rheumatism, either in articular rheumatism, subcutaneous nodules or chorea.

The questions that we should try to answer and the problems that we should attack in this regard are as follows:

Is chronic valvular disease in young people always rheumatic in origin?

In those with chronic valvular disease in youth without a history of rheumatic fever or chorea, has a very mild infection of the sort been overlooked?

Can rheumatic endocarditis occur without any evidence of arthritis or chorea?

Are we frequently or occasionally overlooking the onset of rheumatic endocarditis in very young children?

Is all endocarditis in earliest childhood associated only with rheumatic infection, or may it accompany other types of infection, for example, sepsis, tuberculosis, or some of the lesser diseases of childhood?

Last year, in an effort to contribute a little information in the answers to these questions, Dr. Ledbetter and I made a study of the effect of pertussis on the heart, and failed to find any evidence in a considerable series of cases that valvular disease is produced by such an infection. Scarlet fever has been blamed for the production of chronic valvular disease in a small percentage of cases—about 0.5 of 1 per cent of scarlet fever patients—and such cardiac involvement seems to come with or without complicating arthritis. Further study of the common diseases of childhood is needed, though at the present time such infections as mumps, chickenpox and measles are not considered to be the primary causes of endocarditis. The so-called septic infections of childhood, however, have not been so carefully analyzed as they should be with reference to their effect on the endocardium.

PRESENT OBSERVATIONS

The present paper is, in particular, concerned with the occurrence of endocarditis during the first few years of life. This is obviously of great importance for various reasons. In the first place, children of school age frequently are first discovered to have chronic valvular disease by routine examination by school physicians, when they are 5, 6 or 7 years old, when there is no history of rheumatic fever or chorea.

1. Still G. F.: Endocarditis in Childhood, Common Disorders and Diseases of Childhood, London, H. Frowde, 1912.

Second, it is the child of preschool age who tends to be less carefully examined and followed for mild infections or in a routine way than is his older brother or sister. Third, he is often not able to describe his symptoms when he is ill. Finally, it has been common experience that in the very young child the rheumatic infection tends to be a general, often very vague, disease attacking the heart, but with little or no arthritis or chorea, as MacCallum [2] and Swift [3] have so well pointed out. At necropsy on such subjects one may find the typical Aschoff bodies in the myocardium or endocardium.

One must constantly remember that heart murmurs, even loud ones, do not necessarily mean valvular disease. Acute infection, fever, anemia or ill health otherwise may result in murmurs due to dilatation of the heart from weakness. The results in the subjects reported here have shown either endocarditis at necropsy, or such characteristic murmurs and other signs on examination that there has been no doubt about the diagnosis. It is to be noted, however, that the first sign of mitral valve disease is often an apical systolic murmur. Such a murmur, if intense enough, and in the absence of reason for, or evidence of, cardiac dilatation alone, may mean organic mitral regurgitation. The development of such a murmur under observation, particularly following rheumatic fever or chorea and the frequent addition still later of a mitral diastolic or aortic diastolic murmur, make the diagnosis of clinical mitral regurgitation in some cases quite certain, even though during the last few years there has been a tendency to call all mitral valve disease stenosis. It is true that in the necropsy room mitral regurgitation is almost always associated with at least a slight degree of stenosis, but for practical clinical purposes it is just as true that there is pure mitral regurgitation, pure miral stenosis, and a much more frequent combination of mitral regurgitation and stenosis.[3a] All apical systolic murmurs are certainly not functional, but, of course, good judgment must be used in their interpretation. Patients with early or slight organic mitral regurgitation rarely die; therefore confirmation by necropsy is also rare. Those children with rheumatic heart disease who do die show usually at necropsy cardiac dilatation with or without pericarditis and only rarely mitral stenosis. Of fourteen such cases that came to necropsy at the Boston Children's Hospital from 1915 to 1926 only one child showed mitral stenosis (aged 9 years), while seven had pericarditis. In most of these patients, who were all very sick at the

2. MacCallum, W. G.: Rheumatism: The Harrington Lecture, J. A. M. A. **84**:1545 (May 23) 1925.

3. Swift, H. F.: Rheumatic Fever, Am. J. M. Sc. **170**:631 (Nov.) 1925.

3a. Sometimes this difficulty is circumvented by diagnosing simply "mitral endocarditis" or "mitral valve involvement," but this is not wholly satisfactory when there is marked stenosis or regurgitation.

time of examination, apical systolic murmurs were explained by dilatation with regurgitation through the wide mitral ring. In 4 of these 14 children, however, the mitral valve was deformed, once to cause stenosis, twice with puckering of a cusp to cause regurgitation without stenosis, and once with marked shrivelling of the posterior cusp to cause regurgitation without stenosis. Of 173 children under 12 years of age, seen during the past year in the children's heart clinic at the Massachusetts General Hospital, with definite uncomplicated disease of the mitral valve of "rheumatic" type, only one showed a mitral diastolic murmur alone; 145 showed both systolic and diastolic murmurs, and twenty-nine showed only systolic murmurs.

The observation during the last few years of a few striking cases of beginning valvular disease in earliest childhood has been the chief stimulus of my interest in the subject. The children were seen in the children's heart clinic at the Massachusetts General Hospital and in private practice; they are summarized below. In addition to these cases, notes are given concerning necropsy findings at the Infants' Hospital in Boston and at the Massachusetts General Hospital, and concerning young children with valvular disease seen at the Children's Hospital in Boston during the last year.

CASE REPORTS OF CHILDREN UNDER FIVE YEARS OF AGE WITH VALVULAR HEART DISEASE

CASE 1.—D. B., a boy, aged 21 months, had chorea and mitral endocarditis. His temperature ranged from 98.8 to 99.2 F. The tonsils were large; the heart was enlarged, noted both by physical and by roentgen-ray examination. There was a loud, rough systolic murmur at the apex.

The child had been followed in the feeding clinic since the age of 2 months, having made twenty-three visits. Three months prior to his present illness, the heart was examined and found to be normal. Measles followed by pneumonia occurred two months before the chorea.

A stepbrother also had chorea and endocarditis (case 2).

CASE 2.—S. S., a boy, aged 2 years and 10 months, had mitral regurgitation and stenosis. He came to the hospital for routine examination. The heart was definitely enlarged, and loud blowing systolic and mid-diastolic murmurs were heard at the apex.

Nine months later the boy had chorea. One or two years after the discovery of the heart disease, the murmurs were unchanged. There was no evidence of aortic valve disease.

At the age of 16 months this patient had been in the hospital ward with bronchopneumonia, but four months later (at the age of 20 months) the heart was reported to be normal (outpatient department).

The father of this child had died of heart trouble the year of the patient's birth, and his stepbrother (case 1) later had chorea and mitral endocarditis.

CASE 3.—T. B., a girl, aged 3 years and 10 months, had mitral endocarditis. Four months before examination in the clinic the family physician had discovered the heart disease. Now a very loud, blowing systolic murmur was discovered at the apex. There was little or no cardiac enlargement. There were no symptoms.

During the next two and a half years (up to the time of writing) the condition of the child has been followed. A mitral diastolic murmur has developed

in addition to the systolic murmur. She has had frequent colds, possible chorea, and pertussis without evidence of further cardiac damage.

Two years and three months before the heart lesion was noted, the patient had come to the hospital for treatment of rickets (at the age of 19 months). At that time her heart was normal. There was no past history of rheumatic fever, chorea, scarlet fever or diphtheria. Tonsillitis occurred the year before the heart trouble was found, and her tonsils were removed a year later.

The family history was negative.

CASE 4.—T. O'H., a boy, aged 2½ years, had rheumatic fever. Mitral stenosis and regurgitation resulted. The patient had been ill for four weeks with fever and pain in the knees, elbows and shoulders before entrance to the hospital ward. The knees were slightly enlarged. The heart was slightly enlarged, with a loud, blowing systolic murmur at the apex. The temperature was elevated.

After five weeks in the hospital the patient was discharged as convalescent. On return to the outpatient cardiac clinic, two weeks, two months and three and one-half months later, mitral systolic and diastolic murmurs were well marked, and the heart was moderately enlarged. The child felt very well on these visits and the leukocyte count, which had been 14,000 in the ward, dropped to 9,500.

In the past history there had been only chickenpox.

The family history was negative.

CASE 5.—J. P., a boy, aged 3 years, had mitral stenosis and regurgitation with partial heart block. This patient was referred to the clinic by the family physician because of the heart trouble. The heart showed slight enlargement, a blowing systolic murmur and a well marked mid-diastolic rolling murmur at the apex, with accentuation and reduplication of the pulmonic second sound. There was occasional cardiac arrhythmia. One month later, an electrocardiogram showed partial auriculoventricular block: a P-R interval of 0.35 second with occasional dropped beat.

Six months later, in addition to the mitral murmurs, an aortic diastolic murmur appeared. The heart block continued. There had been two brief fainting spells but otherwise the boy felt well.

In the past, there was pneumonia at the age of 3 months, but at that time the heart was reported to be normal. Pneumonia occurred again when the boy was 2 years old, followed by some "rheumatic" pains in the ankles.

A sister had had both chorea and rheumatic fever.

CASE 6.—R. S., a girl, aged 4 years, had mitral endocarditis. The patient was referred by the family physician because of discovery of heart trouble. There were no symptoms. The heart was moderately enlarged, noted both by physical and by roentgen-ray examination (the latter interpretation stating that the heart was of characteristic mitral shape). There was a loud systolic murmur at the apex. The same observations were recorded at several later visits.

The patient had had german measles, and occasional colds. These were the only infections noted in the past history. The tonsils and adenoids had been removed seventeen months before the heart lesion was observed at examination in the clinic, and at the time of that operation the heart was normal.

An older sister had endocarditis, and has been followed also in the clinic.

CASE 7.—A. T., a boy, aged 2 years and 1 month, had mitral stenosis and regurgitation. This patient was referred to the cardiac clinic from the general medical clinic because of the discovery of heart disease. The heart did not seem to be enlarged, but there were very loud rough systolic and definite mid-diastolic murmurs at the apex. The child looked poorly, and it was thought wise to treat him as a subject with acute endocarditis.

Eighteen months later the child appeared well. The mid-diastolic murmur was still present.

Previous to the finding of the valvular lesion the tonsils had been a source of trouble, and they were removed the day after the heart disease was noted.

One brother, aged 5 years, was said to have endocarditis.

CASE 8.—E. G., a girl, aged 4 years, had rheumatic fever and mitral endocarditis. This child was admitted to the ward with the complaint of rheumatic fever. Immediately after the infection had subsided, a loud, blowing systolic murmur was heard at the apex which persisted at the time of later examination. There was apparently no cardiac enlargement.

In the past there had been no other illnesses.

Two other members of the family had had rheumatic fever.

CASE 9.—W. H., a boy, aged 3½ years, had mitral stenosis. In the course of a routine examination during a study of the families of rheumatic subjects, this boy was found to have a mitral diastolic murmur, with no symptoms.

In the past, he had had measles only. The tonsils were enlarged.

A sister had "rheumatic heart disease."

CASE 10.—T. K., a boy, aged 4 years, had mitral regurgitation and stenosis. This child was referred from the general medical clinic because of the finding of a heart lesion. The heart showed no enlargement, but a short systolic and a mid-diastolic murmur at the apex were present.

The family and past history were negative so far as they could be obtained.

CASE 11.—C. P., a boy, aged 19 months, had mitral endocarditis. In the course of routine examination, this child was discovered to have slight cardiac enlargement and mitral systolic and diastolic murmurs.

There was no history of serious illness in the patient, or of rheumatic infections in the family.

CASE 12.—W. H., a boy, aged 3 years, had rheumatic fever. Mitral regurgitation and stenosis resulted. This child entered the ward with mild rheumatic fever of three weeks' duration. Two weeks after discharge in the cardiac clinic of the dispensary, he showed loud systolic and rumbling diastolic murmurs at the apex without definite cardiac enlargement on physical examination.

In the past, he had had measles and tonsillitis with cervical adenitis at the age of 2 years.

The family history was negative.

CASE 13.—D. D. B., a girl, aged 28 months, had mitral and aortic regurgitation. This child was normal at birth, and, at the age of 22 months, having had a normal heart, gradually developed heart murmurs during the course of the few weeks before I saw her. She was under observation by Dr. M. Sisson. She had had occasional short spells of fever for a few days at a time during the previous six months, apparently "colds," the last time with otitis media. She was quite nervous, but showed no definite chorea, and had never had soreness of joints or muscles. On examination she showed no cardiac enlargement, but a very loud, blowing systolic murmur at the apex and a loud, blowing, early diastolic murmur along the left sternal border. There was also a slight mid-diastolic murmur at the apex. There was no anemia.

The past history (except as before noted) was entirely negative except for pertussis at the age of 1 year.

The family history was negative.

CASE 14.—C. M., aged 4 years, had rheumatic fever, acute endocarditis of mitral and aortic valves, and acute pericarditis. This child entered the ward with fever and malaise of three weeks' duration. The joint symptoms were very mild and there was no note of abnormality of the joints while in the hospital. After two and one-half months in the ward, during which there was evidence of endocarditis and pericarditis, the child died.

Necropsy disclosed acute and chronic endocarditis of the mitral valve, acute endocarditis of the aortic valve, acute and chronic serofibrinous pericarditis and pleuritis, hypertrophy and dilatation of the heart, and chronic passive congestion. The heart weighed 170 Gm.

SUMMARY

In this series of fourteen patients, all with endocarditis and all under 5 years of age, five gave a history of recent chorea or rheumatic fever, another had chorea nine months after the discovery of his heart trouble and still another had had some "rheumatic" pains in the past, following pneumonia. Thus, in seven (or 50 per cent) the condition was quite definitely "rheumatic" in origin. One patient showed valvular disease during colds and otitis media, perhaps coincidentally. The condition in the other six is of more obscure origin, although some of them had had colds or enlarged tonsils. It is possible, but very unlikely, that one or two may have been congenital in origin, or have had no heart disease but simply an unusual functional condition. It is probable that all of these cases including those of unknown etiology were rheumatic in origin, but of this fact we cannot be absolutely certain. The ages (more than two years), the valve lesions, at times the family history, and the well-known preponderance of the rheumatic factor in the etiology of most valvular disease in young people make the diagnosis of rheumatic endocarditis in this entire group of fourteen cases most probable. There was a history of rheumatic fever, chorea, or endocarditis in other members of the family in seven cases.

During the course of the past three years, when all but one of these patients were seen, two other children under 5 years of age were also seen who should be mentioned in this series, even though endocarditis has not as yet been diagnosed definitely. They fall into the group of "potential heart disease" at least.

CASE 15.—A. S., a boy, aged 2½ years, had rheumatic fever. There was a moderate blowing systolic murmur at the apex, without evidence of cardiac enlargement three months later.

CASE 16.—R. R., a girl, aged 3 years, had acute articular rheumatism. She had a second attack of mild rheumatic fever at the age of 3½ years. The heart apparently was not affected.

One of these two cases (the first) gave a positive family history for rheumatic heart disease.

PATIENTS UNDER 5 YEARS OF AGE WITH ENDOCARDITIS, SEEN DURING THE LAST YEAR AT THE CHILDREN'S HOSPITAL IN BOSTON

There were five patients with endocarditis followed during the last year in the cardiac clinic at the Boston Children's Hospital. Four of these had had rheumatic fever and the other one had been treated for "flatfoot" at the age of 21 months. Four were between 3 and 4 years of age, and one between 4 and 5. There were also two other subjects who showed no endocarditis, but who had had rheumatic infections—one rheumatic fever at 3 years of age and the other chorea at 4 years and 10 months. There were six more subjects with endocarditis between the

ages of 5 and 6 years, two of them with definite, one with probable, two with possible and one without rheumatic history. During this year, 228 children were seen in this heart clinic.

NECROPSIES AT THE MASSACHUSETTS GENERAL HOSPITAL

Among 2,000 necropsies in fifteen years at the Massachusetts General Hospital, there were 230 children under the age of 5 years, more than half of whom were less than 1 year old. Of these, only one, aged 4 years, had endocarditis. This child showed rheumatic endocarditis of both mitral and aortic valves and also pericarditis.

There were six cases of pericarditis without endocarditis, one due to acute rheumatic fever, two chronic, and three associated with pneumococcus and staphylococcus infection.

There were eighty-six "septic" cases (pneumonia, meningitis, abscesses, otitis media, septicemia) and in not one of them was there any endocarditis. This shows the rarity of septic endocarditis in early childhood.

Thirty cases of tuberculosis, miliary or pulmonary, showed no endocardial or pericardial involvement, and twenty-three cases of congenital syphilis showed normal hearts and aortas macroscopically.

NECROPSIES AT THE INFANTS' HOSPITAL IN BOSTON

In twelve years (from 1914 to 1926) among almost 7,000 ward patients and 1,276 necropsies, there were ten patients less than 2 years old, in whom the condition was diagnosed as endocarditis; on nine of them necropsies were performed. Seven of these ten endocardial involvements were definitely associated with "septic" infections (pneumonia in four, two of whom also showed pericarditis—one purulent and one serofibrinous; otitis media in one case; purulent tracheobronchitis in one case, and purulent pyelitis in one case). The other three of these ten cases were of uncertain etiology, so far as clinical evidence was concerned; two were associated with gastro-intestinal symptoms, one of these with fever, and the third case with malaise alone. At necropsy, this last-mentioned patient showed rheumatic heart disease with acute endocarditis of the mitral and tricuspid valves, adhesive pericarditis, and Aschoff bodies in both myocardium and endocardium.

Of the nine patients on whom necropsies were performed, the mitral and tricuspid valves were involved in four, the mitral and aortic valves in one, all four valves in one, the mitral valve alone with auricular endocarditis noted in one, and the valves not specified in the remaining two. Of the ten diagnosed as endocarditis loud murmurs were heard in five, while in the other five the heart was essentially normal on physical examination.

At this hospital, during this interval of twelve years, there was only one case of rheumatic fever and none of chorea. The patient with rheumatic fever showed acute pericarditis clinically, and recovered.

CASES AT INFANTS' HOSPITAL

Since the Rotch Memorial Building was opened in March, 1914, the following patients with endocarditis (acquired after birth) or rheumatic fever have been seen:

CASE 1.—E. DeV., a boy, aged 22 months, was seen Aug. 8, 1916. At that time he had rheumatic fever, double otitis media, chronic hypertrophied tonsils and acute pericarditis. There was a systolic murmur at the base of the heart. The patient was discharged relieved. There was a family history of tuberculosis.

CASE 2.—A. S., a boy, aged 4 months, was seen Dec. 18, 1916. There were no murmurs on physical examination. Necropsy showed double pleuropneumonia, acute serofibrinous paricarditis, mitral and auricular endocarditis with vegetations on the mitral valve. The family history was unimportant.

CASE 3.—V. G., a boy, aged 13 days, was seen Dec. 9, 1917. He had tracheobronchitis. Physical examination of the heart was negative. Necropsy showed purulent tracheobronchitis, an enlarged thymus and acute endocarditis. Two young siblings had had colds.

CASE 4.—A. Z., a girl, aged 6 months, was seen Sept. 24, 1917. Lobar pneumonia was present in both lower lobes of the lungs; purulent pericarditis, myocarditis and endocarditis of all valves were also present at necropsy. There were no murmurs on physical examination. The family history was negative.

CASE 5.—I. F. R., a girl, aged 4¼ months, was seen Sept. 22, 1917. Pulmonary atelectasis, otitis media and chronic indigestion from fat and carbohydrates were present. There was a loud systolic murmur transmitted to the left side of the neck and the left axilla. Necropsy showed acute endocarditis of the mitral and tricuspid valves. The family history was negative.

CASE 6.—M. M., a girl, aged 22 months, was seen Nov. 19, 1917. There were no murmurs. Necropsy showed rachitis with multiple fractures, bronchopneumonia, tubular nephritis, definite redness and beading of the mitral and tricuspid valves and marked redness of the aortic valves without thickening. The family history was negative.

CASE 7.—A. M., a girl, aged 7 weeks, was seen Sept. 7, 1918. Indigestion with fermentation, and tubular nephritis were present. Vomiting and diarrhea had been present for three days. She lived for three more days. Examination showed no enlargement of the heart. There was a loud systolic murmur, maximal at the first and second left intercostal spaces. The diagnosis of congenital heart disease with patent ductus arteriosus was made. Necropsy disclosed acute vegetations on the mitral and tricuspid valve, but no congenital lesion. The family history was negative.

CASE 8.—M. B., a girl, aged 14 months, was seen Sept. 28, 1918. Her illness lasted five days, terminating in death. The heart was normal on physical examination. Pyelitis with pus in the kidneys, ureters and bladder was present. Necropsy also showed acute endocarditis, acute myocarditis and dilatation of the right side of the heart. The family history was negative.

CASE 9.—B. La F., a girl, aged 2½ months, was seen Sept. 19, 1918. Congenital pulmonary atelectasis and indigestion from fat were present. The heart was normal on examination. The child died suddenly. Three days before death, she began to have an unexplained slight elevation of temperature. Necropsy disclosed acute endocarditis involving the mitral and aortic valves. The family history was doubtful.

CASE 10.—E. H., a boy, aged 6 months, was seen June 21, 1919. Bronchopneumonia, an abscess on the back and endocarditis were present. There was a history of pneumonia at 2 months, and of otitis media and boils. On examination, the heart was very large with loud systolic and diastolic murmurs everywhere. The roentgenogram showed a large heart, with probable mitral and aortic regurgitation. The child died in five days. Permission for necropsy was not given. The family history was negative.

CASE 11.—A. T. G., a boy, aged 16½ months, was seen June 24, 1922. The family history was negative. The patient had been ill two months prior to examination, with chills and fatigue for one day. One month prior to this he was irritable, sleepless, drowsy, listless and pale, with cough and nightmares; and possibly fever. Three weeks prior to examination, he was weak and unable to stand, and loss of weight was noted. There were no other illnesses. The heart showed a very loud systolic murmur over the entire precordia, loudest at the apex, and was rapid and enlarged. There was a little fever. The child died in four days.

Necropsy showed bronchopneumonia with bilateral pleurisy and central necrosis of the liver, rheumatic carditis (including myocardium), Aschoff bodies in the myocardium and endocardium, acute mitral and tricuspid endocarditis, and adherent pericardium. The heart weighed 167 Gm.

Thus, in nearly 7,000 cases (12,599 minus 5,893) in the last twelve years at the Infants' Hospital, ten cases of endocarditis have been diagnosed, nine proved at necropsy, and one in whom necropsy was not performed. Seven of these were associated with septic infections and three had indefinite conditions consisting in two of the gastro-intestinal symptoms (in one of these there was also fever), and in the third, of malaise. In the last mentioned cases rheumatic heart disease was found at necropsy. One other patient (case 1) had rheumatic fever and acute pericarditis but did not die.

COMMENT

Acute polyarticular rheumatism is rare in infants; endocarditis is less rare but still quite uncommon. It appears from this study that endocarditis in the first two years of life is more likely to be associated with the "septic" type of infections than with the rheumatic, while the reverse holds true after two years of age. It is, therefore, possible that endocarditis may occur in infancy in some cases as the result of septic infections and, if recovery takes place, may later result in valvular deformity and chronic valvular disease of unknown origin. It is more likely that very mild rheumatic infections (including rheumatic heart disease) in young children are occasionally overlooked, accounting for a large percentage of the cases of chronic valvular disease of unknown cause found in children at school ages.

REVIEW OF THE LITERATURE

One of the most important contributions to the subject of endocarditis in earliest childhood has been recently published by Geiger.[4] He

4. Geiger, R.: Endocarditis in Young Children, Ztschr. f. Kinderh. **40**:370 (Dec. 16) 1925.

has reported data on two groups of cases of endocarditis, in all of which the patients were under 4 years of age. The first group is composed of ninety cases selected from the literature; it is not a complete compilation, for most of the cases are those reported by German authorties in publications which were readily obtained by Geiger. They date from 1844 to 1923. The second group consists of eighteen cases seen at the Munich Children's Clinic from 1907 to 1924. Of the first group of ninety cases necropsies were performed on forty-three, and of the second group of eighteen, eleven came to necropsy.

Group 1 consists of thirty cases in the first year of life, eleven in the second, nine in the third and forty in the fourth. The etiologic factors were acute polyarticular rheumatism in twenty cases, chorea in two, and erythema nodosum in one, making a total rheumatic series of 23, or 25.5 per cent. Miscellaneous "septic" infections, such as empyema, accounted for seventeen more cases, pneumonia three, scarlet fever four, tonsillitis six, miliary tuberculosis six (in three, the findings were proved at necropsy), eczema and other skin diseases three, and miscellaneous infections (smallpox and measles, for example) eight. The etiology was obscure in the remaining twenty cases. Fifteen of the cases were diagnosed as septic or ulcerative or malignant endocarditis, in nine of which the observations were proved at necropsy. The right side of the heart was involved in thirteen cases. In only eight cases was the condition chronic.

The most interesting fact relative to this group was that the endocarditis in the youngest patients was more often associated with septic infections than with rheumatism, while in the oldest patients the reverse was true. Of the thirty subjects less than 1 year old, eleven had septic infections and only one was obviously rheumatic; of the forty cases between 3 and 4 years old, only two appeared to be septic in origin and seventeen were rheumatic. This is in accord with my own observations, recorded in the foregoing.

Geiger's second group, the eighteen Munich cases, included three children in the first year of life, one in the second, three in the third and eleven in the fourth. The etiology was analyzed as follows: rheumatic, eight; unknown, six; sepsis (multiple abscesses, furunculosis), three; tonsillitis, one. Those associated with sepsis were all under 2 years of age, while those evidently due to rheumatism were all over 3 years. Necropsies were performed on the four youngest patients in the series, aged 8, 10, 11 and 14 months, respectively; the mitral valve alone was involved in two cases, the mitral and tricuspid valves in one case and the endocardium in one case. The etiology was unknown in one of these four. None of the eleven over 3 years of age was associated with septic infection; eight were rheumatic.

The rarity of endocarditis in infants and its infrequence in children under 5 years of age has been the subject of comment by several writers. Finkelstein,[5] during twenty years in the large clinic of the Children's Hospital in Berlin, found nineteen cases of endocarditis in infants. Steffen [6] reported ten of ninety-five cases of endocarditis to be in children under 4 years of age, and Hochsinger [7] reported six out of fifty-three under 4 years. Gilbert,[8] in 1913, of 114 subjects with acute endocarditis, found four below the age of 4 years. Bass [9] writes: "Of the postnatal infections, one of the youngest cases is that reported by Czerny in an infant, aged 1 month, suffering from septic endocarditis. Such a case, however, is very exceptional, since endocardial inflammation is rare in children under 12 years of age. In 1,000 postmortem examinations Holt found no subject with acute endocarditis under 3 years of age." Northrup,[10] in 1910, reported only one patient with endocarditis under the age of 5 years, seen at the New York Foundling Hospital among 27,278 cases, with between 2,000 and 3,000 necropsies, and that case was malignant or ulcerative. He emphasized the fact that systolic murmurs may be misleading in making the diagnosis. A few writers have made the diagnosis more often. For example Ledford,[11] in 1921, reported sixty-one of 250 children with acute endocarditis under 5 years of age, but none under 2 years.

Rheumatic fever and chorea are even rarer than endocarditis in infants, but after the age of 2 years they become more common. Poynton,[12] in 1918, reported that of 364 cases of rheumatic fever or chorea, first attacks occurred under the age of 1 year in four children, and between 1 and 2 years in three more, while between 2 and 3 years there were thirteen such cases, and between 3 and 4 years, twenty-five. Coombs [13] has reported that of 687 outpatient cases in Bristol with rheumatic heart disease, 7.1 per cent (forty-nine cases) gave evidence

5. Finkelstein, H., in (Endocarditis in Infancy) Lehrbuch der Saüglingskrankheiten, ed. 3, Berlin, 1924.

6. Steffen, J.: Klinik der Kinderkrankheiten (Endocarditis in Childhood) **3**, 1889.

7. Hochsinger, C.: Die Auskultation des kindlichen Herzens, Endocarditis in Childhood, Vienna, 1890.

8. Gilbert, Louis W.: A Study of 197 Cases of Endocarditis in Children at the Massachusetts General Hospital; with Special Reference to the Treatment of Acute Endocarditis, Boston M. & S. J. **169**:85 (July 17) 1913.

9. Bass, M. H.: Endocarditis, chapter 72, Acquired Diseases of the Heart, Abt's Pediatrics **4**, 359.

10. Northrup, William P.: Infrequence of Endocarditis at New York Foundling Hospital, New York, Jacobi-Festschrift, 1910, p. 347.

11. Ledford, H. P.: An Analysis of Two Hundred and Fifty Ward Cases of Acute Endocarditis in Children, Am. J. Dis. Child. **21**:139 (Feb.) 1921.

12. Poynton, F. J.: The Nature and Symptoms of Cardiac Infection in Childhood, Brit. M. J. **1**:417, 1918.

13. Coombs, Carey F.: Rheumatic Heart Disease, New York, 1924.

of active infection within the first five years of life, but practically none of these came within the first three years. In a severe outbreak of acute rheumatism in children in the years 1919 to 1920, Poynton, Paterson and Spence [14] found, out of 172 cases, two that were 2 years old; six, 3 years old and eighteen, 4 years old at the time of the first attack. Rauchfuss,[15] in 1863, reported only two cases in 2,000 nursing infants, and Koplik,[16] addressing this society in 1888, reported only two cases seen among 7,000 sick infants. Miller,[17] in 1899, collected nineteen authentic cases of acute articular rheumatism in infants under 1 year of age, and added another patient of his own. Several individual cases have been reported since, the last recently by Richdorf and Griffith [18] of a case in an infant, aged 6 days, associated with acute rheumatic fever in the mother. Denzer,[19] in 1924, reported three cases of rheumatic heart disease in children under 2 years of age. In children after the second year, as has been noted in the foregoing, particularly by Poynton, typical rheumatic infections are not rare. Of the 185 cases reported by Ingerman and Wilson,[20] two were less than 3 years old and ten were 3 or 4 years of age. Bertram [21] reported no cases of arthritis in infancy in a series of 103, but five cases under 5 years, and five more who were 5 years old. She found no instance of chorea under 3 years of age out of sixty-seven cases. In three cases it developed during the fourth year, and in three during the fifth year. The youngest patient observed in either of her series who showed a definite cardiac lesion was 3½ years old. Finally, in 1908, Poynton and Paine [22] reported observations on sixty-six children under the age of 5 years with first attacks of either arthritis or chorea.

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14. Poynton, F. J.; Paterson, D., and Spence, J. C.: Acute Rheumatism in Children Under Twelve Years of Age, Lancet **2**:1086 (Nov. 27) 1920.

15. Rauchfuss, C.: Acute Rheumatic Polyarthritis in Infants, St. Petersburg med. Wchnschr. **5**:193, 1863.

16. Koplik, H.: Acute Articular Rheumatism in the Nursing Infant, New York M. J. **47**:673, 1888.

17. Miller, D. J. M.: Acute Articular Rheumatism in Infants Under One Year with Report of a Case, Arch. Pediat. **16**:686, 1899.

18. Richdorf, L. F., and Griffith, W. H.: Polyarthritis. Report of a Case in an Infant Six Days Old, Associated with Acute Rheumatic Fever in the Mother, Am. J. Dis. Child. **31**:250 (Feb.) 1926.

19. Denzer, B. S.: Rheumatic Heart Disease in Children Under Two Years of Age, J. A. M. A. **82**:1243 (April 19) 1924.

20. Ingerman, E., and Wilson, M. G.: Rheumatism: Its Manifestations in Childhood Today, J. A. M. A. **82**:759 (March 8) 1924.

21. Bertram, M.: Some Features of the Rheumatic Infection, Brit. M. J. **1**: 497 (March 14) 1925.

22. Poynton, F. J., and Paine, A.: A Contribution to the Subject of Rheumatism Based Upon a Study of Fifty-Two Cases in Children Under Five Years of Age, and an Analysis of One Hundred Cases of Fatal Suppurative Pericarditis in Childhood, Quart. J. Med., April, 1908. Reprinted as Paper No. 21 in Researches on Rheumatism, New York, 1914.

CONCLUSIONS

From a study of the literature, and of cases at the Massachusetts General Hospital, Children's and Infants' Hospitals of Boston, and in private practice, the following conclusions have been reached.

1. Endocarditis in infancy is rare, but even during the first year of life it may occur, and when it does it appears to be more often associated with infection of "septic" nature rather than with rheumatic infection.

2. Acute articular rheumatism and chorea are very rare in infancy, but increase progressively in frequency after the age of 2 years. It is to be remembered that often very mild rheumatic infections and even rheumatic carditis alone may occur in early childhood.

3. Endocarditis after the age of 2 years becomes more common, and is much more likely to be associated with rheumatic rather than with septic infections as the child grows older.

4. Many cases of endocarditis in early childhood are of unknown cause, and doubtless account for much of the chronic valvular disease of obscure etiology found in older children at school age and after, and in young adults. It is probable that the largest percentage of these doubtful cases results from unrecognized rheumatic infection, often very mild, and sometimes affecting the heart alone. It is possible that some of the doubtful cases may result from septic infection in infancy or afterward.

5. Careful observation of the condition of the heart at birth, and during and after every infection in early childhood, is an important measure in the collection of data for the analysis of the large group of cases of endocarditis of unknown origin. Even a slight rise of the temperature above normal or unexplained malaise may mean endocarditis in a child, particularly in the climate of northeastern United States where rheumatic infections are so common and so serious, especially in crowded damp homes in the winter and spring. The presence or absence of a family history of rheumatic infections should always be determined; it appears to be of considerable importance.

6. It is possible that in some cases endocarditis may develop in children very insidiously, with no evident infection to call attention even to the need of medical advice. Routine physical examination, such as is now becoming the practice in the case of many adults annually, would detect such endocarditis in its early stages.

7. Young children with rheumatic endocarditis rarely die, and so necropsy findings revealing early or relatively slight mitral valve involvement are rare. Clinically one finds the loud systolic murmur of mitral regurgitation often preceding the diastolic murmurs of mitral stenosis and of aortic regurgitation in date of discovery.

CALCIUM BALANCE WITH HYDROCHLORIC ACID MILK*

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During the past three years, we have been feeding the rachitic infants in St. Mary's Hospital on hydrochloric acid milk. We have observed that the rachitic bones in these infants healed much faster than when any other formula was used. During this time also we have fed routinely over 325 normal infants on hydrochloric acid milk throughout the first year and have never seen a case of rickets develop in this group.

Because of these observations, we decided to study the calcium metabolism in both rachitic and normal infants fed on sweet milk and acidified milk mixtures.

L. E. Holt, Jr.,[1] and his co-workers showed that the rachitic infant is unable to retain much of his calcium intake, but when cod liver oil is added to his dietary the calcium retention rapidly increases. We wished to see if we could bring about the same results merely by giving the rachitic infant hydrochloric acid milk without cod liver oil, and also to observe what effect the acidified milk would have on the calcium balance of a normal infant.

PROCEDURE

Our plan of procedure was first to put the infant on a sweet milk mixture without cod liver oil for three days, and then measure the calcium intake and output for the next forty-eight hours. Following this, the infant was given the acidified milk for the same length of time, also without cod liver oil, and the calcium retention measured for the ensuing two days.

Our infants were divided into two groups: (1) normal babies, and (2) rachitic infants. The segregation was made by physical and roentgenologic examinations. The severity of the rickets was designated as severe, moderate and mild.

In each case the infant was admitted to the hospital for an examination period of ten days. After he had been on the designated formula for three days, he was placed on a hammock metabolism frame and the experiment started.

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* Received for publication, June 24, 1926.

* From the Childrens' Service, St. Mary's Hospital.

1. Holt, L. E., Jr.; Orr, W. J.; Wilkins, Lawson; and Boone, F. H.: The Calcium and Phosphorus Metabolism in Rickets, Am. J. Dis. Child. **26**:363 (Oct.) 1923.

He was given 0.3 Gm. of carmine, followed immediately by the formula used. During the next forty-eight hours, all the stools and the urine were collected. At the end of this period he was given 0.5 Gm. of powdered charcoal. When this appeared in the stools he was removed from the metabolism frame and the experiment was brought to an end.

The calcium in the stools and urine was determined by McCrudden's method. The calcium intake was measured by determining the calcium monoxide in a 100 cc. sample of the formula, and the total consumption estimated from the quantity consumed during the forty-eight hour period.

In each case the infant was first given a sweet milk formula of whole milk, 960 cc.; water, 240 cc.; dextrimaltose, 60 Gm., or plain certified milk if over 1 year of age.

Following the first metabolism test the same infant was put on whole milk, 960 cc.; tenth normal hydrochloric acid, 240 cc.; dextrimaltose, 60 Gm., or on whole milk, 1,200 cc.; normal hydrochloric acid, 30 cc., depending on the age of the infant.

In this manner the infant had the same fat intake with both the sweet milk and the acidified mixtures. This obviated any variation in calcium absorption due to variations of fat consumption, a factor pointed out by Holt, Courtney and Fales.[2]

RESULTS

An examination of the tabulated results will show that there is no variation of the calcium retention for normal infants when sweet or acidified milk was used.

On the other hand, the calcium retention of the rachitic infant on sweet milk was much less than when hydrochloric acid milk was employed. The more severe the rickets, the greater was this difference. In short, with the aid of hydrochloric acid milk, the rachitic infant has almost a normal calcium retention.

TABLE 1.—*Normal Baby*

Baby W.; aged 10 months; weight, 8,100 Gm.

Type of Formula	Total Intake, Cc.	Gm. CaO in 100 Cc. Formula	Total CaO Intake, Gm.	Urine Output, 24 Hours, Gm.	Total CaO in Urine, Gm.	Weight Dried Stools, Gm.	Total CaO in Stools, Gm.	Total CaO Output, Gm.	Total CaO Retention, Gm.	CaO Retention, per Cent
Sweet milk (milk, 32 oz.; water, 8 oz.; dextrimaltose, 2 oz.)	2,520	0.1493	3.7626	852	0.0258	10.0122	0.8206	0.8464	2.6562	72.1
Acidified milk (milk, 32 oz.; tenth normal hydrochloric acid, 8 oz.; dextrimaltose, 2 oz.)	2,340	0.1462	3.4768	1,120	0.0314	8.9948	0.8632	0.8946	2.5822	74.2

2. Holt, L. E.; Courtney, Angelia; and Fales, Helen L.: Calcium Metabolism of Infants and Young Children, Am. J. Dis. Child. **19**:97 (Feb.) 1920.

TABLE 2.—*Normal Baby*

Baby B.; aged 11 months; weight, 9,910 Gm.

Type of Formula	Total Intake, Cc.	Gm. CaO in 100 Cc. Formula	Total CaO Intake, Gm.	Urine Output, 24 Hours, Gm.	Total CaO in Urine, Gm.	Weight Dried Stools, Gm.	Total CaO in Stools, Gm.	Total CaO Output, Gm.	Total CaO Retention, Gm.	CaO Retention, per Cent
Sweet milk (certified milk).....	2,580	0.159	4.1022	520	0.0232	8.4802	0.8032	0.8364	3.2658	78.6
Acidified milk (certified milk, 40 oz.; normal hydrochloric acid, 1 oz.).....	2,880	0.162	4.6656	1,340	0.0322	8.8602	0.9602	0.9924	3.6732	78.7

TABLE 3.—*Very Slight Rickets*

Baby K.; aged 8 months; weight, 8,595 Gm.

Type of Formula	Total Intake, Cc.	Gm. CaO in 100 Cc. Formula	Total CaO Intake, Gm.	Urine Output, 24 Hours, Gm.	Total CaO in Urine, Gm.	Weight Dried Stools, Gm.	Total CaO in Stools, Gm.	Total CaO Output, Gm.	Total CaO Retention, Gm.	CaO Retention, per Cent
Sweet milk (milk, 32 oz.; water, 8 oz.; dextrimaltose, 2 oz.)	1,740	0.1232	2.1436	500	0.0216	7.0002	0.7532	0.7748	1.3688	63.9
Acidified milk (milk, 32 oz.; tenth normal hydrochloric acid, 8 oz.; dextrimaltose, 2 oz.).....	2,520	0.1301	3.2784	1,060	0.0420	8.4000	0.8654	0.9074	2.3710	72.4

TABLE 4.—*Moderate Rickets*

Baby P. B.; aged 8 months; weight, 6,750 Gm.

Type of Formula	Total Intake, Cc.	Gm. CaO in 100 Cc. Formula	Total CaO Intake, Gm.	Urine Output, 24 Hours, Gm.	Total CaO in Urine, Gm.	Weight Dried Stools, Gm.	Total CaO in Stools, Gm.	Total CaO Output, Gm.	Total CaO Retention, Gm.	CaO Retention, per Cent
Sweet milk (milk, 32 oz.; water, 8 oz.; dextrimaltose, 2 oz.)	2,220	0.1280	2.8416	832	0.0380	9.1200	1.2786	1.3166	1.5250	50
Acidified milk (milk, 32 oz.; tenth normal hydrochloric acid, 8 oz.; dextrimaltose, 2 oz.).....	2,220	0.1280	2.8416	960	0.0430	10.8400	0.7968	0.8398	2.0018	70

TABLE 5.—*Severe Rickets*

Baby J. G.; aged 12 months; weight, 8,100 Gm.

Type of Formula	Total Intake, Cc.	Gm. CaO in 100 Cc. Formula	Total CaO Intake, Gm.	Urine Output, 24 Hours, Gm.	Total CaO in Urine, Gm.	Weight Dried Stools, Gm.	Total CaO in Stools, Gm.	Total CaO Output, Gm.	Total CaO Retention, Gm.	CaO Retention, per Cent
Sweet milk (certified milk).....	1,500	0.1650	2.5740	650	0.0334	20.2134	1.7884	1.6218	0.9522	30
Acidified milk (certified milk, 49 oz.; normal hydrochloric acid, 1 oz.).....	1,380	0.1600	2.2086	808	0.0582	11.2400	0.4306	0.4882	1.7198	77

COMMENT

It is evident from the foregoing that hydrochloric acid milk favors the absorption of calcium in the rachitic infant, but has little effect on the calcium retention of the normal baby.

The reasons for this seem twofold: First, the addition of hydrochloric acid to cow's milk has made the calcium more accessible for absorption. Much of the calcium in cow's milk is found in an insoluble calcium casein. This is converted to soluble calcium chloride by the hydrochloric acid. Zotterman [3] demonstrated this when he showed that by adding hydrochloric acid to cow's milk he could increase the diffusible calcium 400 per cent.

The other factor seems to be the moderate hypochlorhydria which is present in the rachitic and tetanic infant, as pointed out by Babbott.[4] Because of the high buffer content of cow's milk, the rachitic infant has difficulty in first saturating its buffers, and then converting the insoluble calcium to soluble calcium chloride. This the normal infant can readily accomplish, as he has a rather elastic secretory zone for hydrochloric acid in his stomach.

The prophylactic action of hydrochloric acid milk against rickets seems to be due to three factors: First, because of the low buffer contents of hydrochloric acid milk, we can give young infants a high percentage of fat which is readily handled. Fat is probably the vehicle of the antirachitic factor. Hence by increasing the fat consumption we increase the intake of the antirachitic factor. Second, this type of feeding insures a high absorption of calcium. De Buys and von Meysenbug,[5] studying the incidence of rickets in breast fed infants, showed that those infants fed on breast milk low in calcium developed rickets. Third, in most acute or chronic infections there is a reduction of the hydrochloric acid secretory power of the stomach. It is a well established fact that infants suffering from chronic infections, such as pyelitis, colitis, etc., are especially prone to rickets. These infants suffer from a moderate hypochlohydria during the course of their infection, with the result that they have not sufficient hydrochloric acid to saturate the buffers and convert the insoluble calcium salts. This they can accomplish with acidified milk, which insures them a sufficient calcium absorption during their illness.

SUMMARY

1. Hydrochloric acid milk increases the retention of calcium chloride in the rachitic infant, but has no effect on the calcium metabolism of the normal baby.

2. Hydrochloric acid milk has some prophylactic action against rickets during the first year, as it insures a high constant calcium absorption during this time.

3. Zotterman: Acta Pediat. **5**:190, 1923.

4. Babbott, F. L., Jr., and others: Hydrogen Ion Concentration of Gastric Contents of Infants, Am. J. Dis. Child. **26**:486 (Nov.) 1923.

5. De Buys, L. R., and von Meysenbug, L.: The Calcium Content of Breast Milk in Relation to Rickets, Am. J. Dis. Child. **27**:438 (May) 1924.

GROWTH STANDARDS

HEIGHT, CHEST-GIRTH AND WEIGHT FOR PRIVATE SCHOOL BOYS *

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Although for public school work the best standards are those published by Baldwin,[1] we believe that separate standards are necessary for judging the development of boarding and country day school boys; excluding (*a*) private schools in the city; (*b*) boys substandard to physical examination; (*c*) boys whose parents may be known to be of

TABLE 1.—*Height, Chest, Weight for Age, for Private School Boys*

Age, Nearest Year	Height (Net)		Chest Girth (Net) Nipple Level, Midphase		Weight (Net)	
	Cm.	Inches	Cm.	Inches	Kg.	Pounds
6	119.0	46.9	57.9	22.8	19.2	42.4
7	124.1	48.8	59.9	23.6	23.5	51.9
8	130.2	51.3	62.5	24.6	26.1	57.6
9	135.5	53.4	64.5	25.4	29.2	64.4
10	139.8	55.0	66.3	26.1	32.2	70.9
11	142.7	56.2	68.3	29.9	33.7	74.3
12	149.2	58.6	70.3	27.7	38.7	85.3
13	152.8	60.1	72.6	28.6	41.4	91.2
14	162.0	63.8	76.2	30.0	49.4	108.8
15	167.5	65.9	81.0	31.9	54.1	119.2
16	172.2	67.8	85.1	33.5	59.0	130.0
17	174.5	68.7	86.9	34.2	61.1	134.6
18	176.7	69.6	89.9	35.4	65.5	144.4
19	176.9	69.7	90.9	35.8	66.6	146.8
20	177.8	70.0	....	....	66.5	146.5

European stock; (*d*) boys measured by observers or with apparatus whose accuracy was, on investigation, felt to be doubtful.

Such standards have been offered previously, based on 380 boys in Groton, Middlesex and Rivers' schools.[2] That basis is here enlarged to a total of 1,016 by the consolidation of records on 370 boys in the Protestant Episcopal Academy, measured by one of us (F. F.), and

* Received for publication, June 10, 1926.

* From the Santa Barbara Clinic.

1. Baldwin, B. T.: The Use and Abuse of Weight-Height-Age Tables as Indexes of Health and Nutrition, J. A. M. A. **82**:1 (Jan. 5) 1924; Am. J. Phys. Anthrop. **8**:1 (Jan.-March) 1925.

2. Gray, H., and Jacomb, W. J.: Size and Weight in One Hundred and Thirty-Six Boarding School Boys, Am. J. Dis. Child. **22**:259 and 272 (Sept.) 1921; Gray, H., and Edmands, G. H.: Indices of the State of Nutrition in Children, ibid. **23**:226 (March) 1922.

266 boys in the Hill School, measured by Dr. S. T. Nicholson, with whom we plan to publish a study on variability (coefficient of variation).

The present tabulations confirm and smooth our earlier tentative standards. They exceed in stature and chest-girth those reported recently by Baldwin [3] in his monograph on California gifted children, as being "above the best standards for American born children in physical

TABLE 2.—*Weight for Height Standard, for Private School Boys*

Height (Net)		Weight (Net)		Height (Net)		Weight (Net)	
Cm.	Inches	Kg.	Pounds	Cm.	Inches	Kg.	Pounds
111	43.7	19.0	41.9	152	59.8	40.6	89.5
112	44.1	19.3	42.5	153	60.2	41.4	91.3
113	44.5	19.6	43.2	154	60.6	42.2	93.0
114	44.9	20.0	44.0	155	61.0	43.0	94.8
115	45.3	20.2	44.6	156	61.4	43.8	96.6
116	45.7	20.5	45.3	157	61.8	44.6	98.3
117	46.1	20.9	46.1	158	62.2	45.6	100.5
118	46.5	21.3	46.9	159	62.6	46.5	102.5
119	46.9	21.7	47.8	160	63.0	47.5	104.7
120	47.2	22.0	48.5	161	63.4	48.6	107.2
121	47.6	22.4	49.4	162	63.8	49.9	109.9
122	48.0	22.8	50.2	163	64.2	51.0	112.4
123	48.4	23.2	51.1	164	64.6	52.0	114.6
124	48.8	23.6	52.0	165	65.0	52.9	116.6
125	49.2	24.1	53.1	166	65.4	53.8	118.6
126	49.6	24.6	54.2	167	65.7	54.7	120.6
127	50.0	25.0	55.1	168	66.1	55.6	122.6
128	50.4	25.5	56.2	169	66.5	56.5	124.5
129	50.8	26.1	57.5	170	66.9	57.3	126.4
130	51.2	26.5	58.4	171	67.3	58.2	128.3
131	51.6	27.1	59.7	172	67.7	59.3	130.6
132	52.0	27.6	60.8	173	68.1	60.2	132.8
133	52.4	28.2	62.2	174	68.5	61.3	134.8
134	52.8	28.7	63.3	175	68.9	62.1	137.0
135	53.2	29.3	64.6	176	69.3	63.0	139.0
136	53.5	29.8	65.7	177	69.7	63.9	140.9
137	53.9	30.3	66.8	178	70.1	64.6	142.5
138	54.3	30.9	68.1	179	70.5	65.5	144.5
139	54.7	31.6	69.7	180	70.9	66.4	146.3
140	55.1	32.2	71.0	181	71.3	67.2	148.1
141	55.5	32.7	72.1	182	71.7	68.0	150.0
142	55.9	33.3	73.4	183	72.0	68.8	151.7
143	56.3	34.0	75.0	184	72.4	69.6	153.5
144	56.7	34.7	76.5	185	72.8	70.4	155.1
145	57.1	35.3	77.8	186	73.2	71.1	156.8
146	57.5	36.0	79.4	187	73.6	71.9	158.5
147	57.9	36.7	80.9	188	74.0	72.6	160.0
148	58.3	37.5	82.7	189	74.4	73.2	161.4
149	58.7	38.2	84.2	190	74.8	73.7	162.5
150	59.1	39.0	86.0	191	75.2	74.2	163.5
151	59.4	39.7	87.5	192	75.6	74.6	164.4

growth status." In weight for height, on the other hand, they present a strikingly slender type.

To judge a boy's height or chest-girth, one looks in table 1. To judge weight one uses (not the last columns in table 1, which are tabulated merely in case of comparison with older tables), but table 2.

3. Baldwin, B. T.: Anthropometric Measurements, in Terman and Others' Mental and Physical Traits of a Thousand Gifted Children, Genetic Studies of Genius, vol. I, Palo Alto, Stanford Univ. Press, 1925, p. 135.

PROGRESS IN PEDIATRICS

THE PRACTICAL VALUE OF BIOLOGIC THERAPY IN THE PROPHYLAXIS AND TREATMENT OF SCARLET FEVER *

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Since the Schick test has proved so reliable for determining susceptibility to diphtheria, and toxin-antitoxin mixtures have been so successful in vaccination against the disease—to say nothing of the value of diphtheria antitoxin in passive prophylaxis and treatment—it is no wonder that similar attempts have been made in the specific prophylaxis and treatment of scarlet fever. The Dicks discovered three years ago that the disease apparently was caused by a hemolytic streptococcus and its soluble or exogenous toxin. A large amount of investigation has been devoted to the skin test for susceptibility to scarlet fever, to vaccination with streptococcus toxin and to prevention and treatment with antiscarlet fever streptococcus serum. It is my purpose to summarize briefly their practical value and applications at the present time. I fully realize that our information is as yet quite incomplete in these subjects, and especially in relation to vaccination against the disease with subcutaneous injections of the toxin.

IS STREPTOCOCCUS SCARLATINAE THE CAUSE OF SCARLET FEVER?

But before considering these subjects it may be asked whether *Streptococcus scarlatinae* (Dick) has been definitely proved the cause of scarlet fever. That streptococci bear an important relation to the disease and especially in the production of its angina, adenitis and other complications has been long accepted but until the investigations of the Dicks [1] in 1923 it was generally agreed that while the streptococcus was an important secondary organism it could not be regarded as the actual primary etiologic agent. Since then not a few have questioned the evidence, especially Di Cristina [2] and his colleagues in Italy. Unfortunately no way has yet been found to produce the disease experimentally in the lower animals or quickly to differentiate *Streptococcus scarlatinae* from other streptococci in the laboratory in the same way that the

* Received for publication, June 12, 1926.

* Read by invitation before the American Child Health Association, May 19, 1926.

1. Dick, G. F., and Dick, G. H.: Experimental Scarlet Fever, J. A. M. A. **81**:1166 (Oct. 6) 1923.

2. Di Cristina, reviewed by Porter: Arch. Pediat. **43**:197, 1926.

bacteriologic diagnosis of diphtheria is made. The following facts may be accepted, however, as indicating that a toxin producing streptococcus is the primary etiologic agent of scarlet fever and that vaccination and treatment with it and its products is on a rational basis:

1. The production of scarlet fever-like reactions in human beings following the subcutaneous injection of streptococcus vaccines by Gabritschewsky and others in Russia.

2. The production of scarlet fever among volunteers by injections of streptococcus toxin by the Dicks. This, I believe, has been the most convincing evidence of all. It is eminently fitting in passing to pay tribute to the bravery of these volunteers, whose act ranks with that of those who risked their lives in the yellow fever investigation of many years ago, and to express the hope that a more fitting way may be found to record permanently their contribution to medical science and human welfare.

3. The occasional production of scarlet fever-like reactions among those who have received a first injection of 500 or more skin doses of the toxin in vaccination.

4. The extinction of the Dick skin toxin reaction during and following scarlet fever which indicates the production of a specific antibody during the disease for this streptococcus toxin.

But do these facts conclusively prove that a specific streptococcus is the primary etiologic agent of the disease or do they but furnish additional evidence of the importance of streptococci as organisms of secondary infection? That streptococci of apparently nonscarlatinal origin may produce scarlet fever-like exanthems cannot be denied since these are sometimes seen in surgical infections and especially after burns. In other words, the toxins of some strains of virulent *Streptococcus pyogenes* are capable of producing toxic rashes, but in my opinion these need not influence us against accepting *Streptococcus scarlatinae-hemolyticus* as the specific cause of scarlet fever. This seems true especially because the antibody produced during the disease is apparently specific for its toxins and because Moser,[3] the Dicks, and Dochez [4] have been able to prepare immune serums of specific prophylactic and curative properties which are also capable of neutralizing the toxin in the skin, as determined by the blanching test of Schultz and Charlton.

PRACTICAL VALUE OF THE DICK TOXIN SKIN TEST

Is the Dick skin test reliable and acceptable as a means for determining susceptibility to scarlet fever? Is it applicable on a broad scale in a manner analogous to the utilization of the Schick test for determining

3. Moser: Wien. klin. Wchnschr. **15**:1053, 1902.
4. Dochez: Proc. Soc. Exper. Biol. & Med. **4**:184, 1924.

immunity in diphtheria. Has it any value in aiding the diagnosis of clinically doubtful cases of scarlet fever?

In my experience no one has yet developed scarlet fever among those yielding negative reactions, but it would appear that a negative Dick test as applied under routine conditions is not quite as reliable as an index of immunity as a negative Schick reaction under similar conditions. The toxins of different strains of *Streptococcus scarlatinae* may yield slightly varying results and when one toxin is being employed it should represent a mixture of two or more strains. The weakly positive reactions are somewhat difficult to interpret and I agree with Lindsay, Rice and Selinger [5] that a slightly larger dose of toxin should be used in conducting the test in order to detect those who are probably susceptible despite the presence of a small amount of antitoxin in the blood. In other words, the present day difficulties with the skin test are largely of a technical nature. By using a polyvalent toxin that has been adequately tested in a group of known susceptible and immune persons, in slightly higher concentration than usual and making the readings in from eighteen to twenty-four hours after injection, the test can be made satisfactory and acceptable for determining susceptibility to scarlet fever.

Among infants of from 1 to 6 months of age the skin test is hardly worth while. From 40 to 50 per cent are apparently susceptible and yield positive reactions, and practically 25 per cent of those immune and negative reactors are almost sure to become susceptible by the time they reach 3 years of age. Approximately 60 per cent of children from 1 to 5 years of age yield positive reactions, and if active immunization is ever placed on a sound basis it is highly probable that the skin test will be dispensed with as a routine procedure and all children of preschool age immunized, as is the present tendency with toxin-antitoxin immunization in diphtheria. After 5 years of age, however, the skin test is advisable as preliminary to active immunization, since the percentage of positive reactions drops to the neighborhood of 30 per cent for the group between 5 and 10 years, 20 per cent for from 10 to 15 years, and 15 per cent among adults. In other words, both in private and in institutional practice the skin test is unreliable among infants under 6 months of age because a negative reaction means little or nothing, since the immunity may readily disappear within the following year. It might be worth while between 6 months and 5 years because approximately 35 per cent are immune, and it is decidedly worth while after 5 years of age because from 60 to 85 per cent are apparently sufficiently immune to escape infection. Furthermore, the size of a positive reac-

5. Lindsay, J. W.; Rice, E. C., and Selinger, M. A.: Scarlet Fever, J. A. M. A. **86**:1191 (April 17) 1926.

tion is somewhat of an index to the size of the first dose of toxin if vaccination is to be done, since a strongly positive reaction in my opinion indicates that the first dose of toxin should not be more than 250 skin doses. This is in order to guard against producing a marked systemic reaction.

May a child or adult possessing natural immunity and yielding a negative skin reaction be safely regarded as immune for the balance of life? A final answer cannot be given at present to this important question but the chances are quite good that such is the case. Subjects have occasionally yielded positive reactions in my experience after former negative reactions, but I could not be certain whether these were due to a disappearance of natural immunity or to the use of defective toxins. But it is certain that the immunity following active toxin immunization may wear away and that a person yielding a negative reaction after vaccination may later react positively. But whenever the skin test with a reliable toxin yields a negative reaction in a subject after the first year of age, I think we may safely conclude that immunity sufficient for protection against scarlet fever is present.

Furthermore, the test is of some value in differential diagnosis when applied *early*, especially in surgical cases and burns, because a positive reaction indicates that the rash *may* be scarlatinal, while a negative reaction indicates that it could not be since it is indicative of the presence of immunity.

PRACTICAL VALUE OF TOXIN VACCINATION

A subject of more importance, however, is that of vaccination against scarlet fever, since approximately 45 per cent of children from 1 to 15 years of age and 15 per cent of adults are believed to be susceptible on the basis of the skin test, while the mortality under usual treatment without serum is in the neighborhood of 25 per cent for children of from 6 months to 5 years and from 3 to 4 per cent after this age. This makes no mention of the from 3 to 30 per cent of such crippling complications as nephritis, adenitis, otitis media and mastoiditis. With the very satisfactory status of toxin-antitoxin immunization in diphtheria before us, it was and still is only natural to hope that a similar state of affairs may be reached in the prevention of scarlet fever.

But it does not appear that the toxin of *Streptococcus scarlatinae* is as antigenic as the toxin of the diphtheria bacillus; certainly it is somewhat more difficult to produce antibody with it in both human beings and the lower animals and the resulting immunity does not appear to be of as long duration.

The doses first advised are now known to be inadequate, and no one at present knows just what the doses should be for the best results under routine conditions. Approximately a total of from 1,500 to 3,000 skin test doses of diphtheria toxin divided into the usual three injections

and protected with an adequate amount of antitoxin, are sufficient for producing immunity in diphtheria. These amounts of streptococcus toxin have not, however, yielded satisfactory results in vaccination against scarlet fever. At the present time the Scarlet Fever Committee recommends five injections of 500, 1,500, 5,000, 15,000 and 20,000 skin test doses of toxin at intervals of a week as a minimum. While it is usually possible to give these in private and institutional practice, in which cooperation is good, difficulties are presented in public health work on a broad scale. For this reason the recent recommendation of Young and Orr [6] of three injections of 500, 5,000 and 30,000 skin test doses at intervals of two weeks, is worthy of serious consideration. At all events it would appear that a total large dosage is required, while at the same time it is necessary to avoid systemic reactions by starting with not more than from 250 to 500 skin test doses for the first injection. It may be possible to avoid toxic reactions by combining a small amount of scarlet fever antitoxin with the toxin after the manner of the T-A mixtures in diphtheria vaccination, but the results of some experiments along these lines have not been satisfactory. The methods of detoxifying the toxin with sodium ricinoleate by the method of Larson, Huenekens and Colby [7] are still under trial, with somewhat discouraging preliminary results.

Active immunization against scarlet fever is therefore still in the experimental stage and especially as a public health measure on a large scale. In private and institutional practice, however, I think that enough has been done to show that the total injection of at least 20,000 skin test doses of toxin in five injections at intervals of one week confers an immunity in at least from 80 to 90 per cent of positive skin reactors. My present practice is to administer the doses recommended by the Scarlet Fever Committee, except that the first one is reduced to 250 skin test doses in the cases of children and adults giving strongly positive preliminary skin reactions. No one can state how long the immunity lasts, but it is apparently safe to predict at least three years for the majority and I believe that the interval will be much longer after immunization with these large amounts of toxin. It is true that five injections may be a nuisance for some. The injections are ordinarily well borne, however, anaphylaxis is quite rare and the inconvenience quite worth while, and especially in institutions for children and in private practice in the presence of epidemics. In my experience susceptibility to scarlet fever does not appear to be temporarily increased by the production of a negative phase of temporary depression of resistance when the first dose does not exceed 500 skin test doses. In other words,

6. Young, C. C., and Orr, P. F.: Dosage of Toxin for Active Immunization Against Scarlet Fever, J. A. M. A. **86**:1340 (May 1) 1926.

7. Larson, W. P.; Huenekens, E. J., and Colby, Woodward: Immunization Against Scarlet Fever, J. A. M. A. **86**:1000 (April 3) 1926.

I believe physicians do well to conduct the preliminary skin test and vaccinate those with positive reactions or susceptible in families and institutions, although the method is not yet ready for wholesale application in schools and communities because of the uncertainty regarding total dosage, number of injections, reactions and duration of immunity. Whether or not vaccination with streptococcus vaccine as practiced by Savchenko [8] and Gabritschewsky [9] as long as twenty years ago in Russia or with mixtures of vaccine and toxin are superior to the toxin alone cannot be stated. In 1912, however, I [10] thought vaccination with vaccine decidedly worth while and now greatly regret that I did not continue my investigation for more conclusive results.

PROPHYLAXIS BY ANTISCARLET FEVER SERUM

Fortunately the immunity following injections of the toxin develops much more quickly than is the case in diphtheria vaccination with toxin-antitoxin but not quickly enough to afford protection during the incubation period of scarlet fever or after intimate exposure to the disease. Data at hand indicate, however, that immediate protection may be secured by the subcutaneous or intramuscular injection of a dose of antiscarlet fever serum sufficient for neutralizing from 50,000 to 100,000 skin test doses of toxin. With highly concentrated serum this may amount to the injection of from 1 to 5 cc.

Under conditions of intimate exposure this passive immunization is to be recommended and especially in cases that have reached the end of the period of incubation, but the injection is always likely to sensitize the subject to horse serum and serum sickness follows in a large percentage of persons. There is usually time for conducting a preliminary Dick toxin skin test, and this is my practice in order to avoid the needless administration of serum. In this connection it is to be hoped that our commercial laboratories will render available an immune serum for prophylactic purposes prepared by the immunization of goats or cattle, just as I believe and have long advocated the preparation of diphtheria and tetanus antitoxins for prophylactic purposes by the immunization of cattle in order to reduce sensitization to horse antiserum reserved for use in the treatment of these diseases. Even though such an antiscarlet fever serum is not available at present and the injection of the present serums may produce sensitization and disagreeable serum sickness, yet when this is compared to the dangers of an attack of scarlet fever I believe it sensible and advisable to administer them for prophylactic purposes in the presence of intimate exposure in private families or in

8. Savchenko: Russk. Vrach. **25**:797, 1905.
9. Gabritschewsky: Russk. Vrach. **4**:941, 1905; ibid. **10**:469, 1906.
10. Kolmer: Pennsylvania M. J. February, 1912.

institutional outbreaks of the disease. An immunity is apparently produced in most persons which appears to last for at least from four to six weeks, as judged by the results of skin tests.

CURATIVE VALUE OF ANTISTREPTOCOCCUS SERUM

Certainly the curative value of antiscarlet fever serum prepared by either the Dick or Dochez methods, or a combination of these, appears to have been definitely established. Indeed, I believe it may be stated that the administration of this serum should constitute a part of the treatment of all cases just as antitoxin is used in a routine way in the treatment of diphtheria. It may be that the serum could be omitted in the treatment of the mild cases in order to avoid sensitization and the production of serum sickness, which follows in a large percentage of cases, but in my opinion this is inadvisable; mild cases may become severe and develop the complications of nephritis, adenitis and otitis media, which are much more dangerous than serum disease and usually prevented by the early administration of serum in adequate dosage. For these reasons I believe that the immediate subcutaneous or intramuscular injection of a dose of concentrated serum sufficient for neutralizing at least 200,000 skin test doses of toxin should be a routine practice, followed by subsequent injections if necessary as indicated by the temperature, rash and mental state of the patient. In severe infections and when the serum is given after the second to fourth days of the disease, it is sometimes advisable to give the serum by intravenous injection. If this is impossible or considered dangerous and inadvisable, however, the intramuscular route is to be preferred to the subcutaneous; it is just as easy and more efficacious because of quicker absorption.

The prompt administration of serum has certainly reduced the mortality and the incidence of complications even though it has little or no curative influence on such complications as otitis, mastoiditis and adenitis when these have developed. In cases of bacteremia, however, with the streptococcus in the blood as detected by blood culture, I believe that intravenous or intramuscular injections of serum should be employed and in these my preference is for the Dochez antibacterial serum or one prepared by immunizing horses with both the toxin and streptococci.

DIAGNOSTIC VALUE OF THE SCHULTZ-CHARLTON REACTION

Finally brief mention may be made of the diagnostic value of injecting of 0.1 cc. of antiscarlet fever serum *into* the skin presenting a rash. As shown by Schultz and Charlton,[11] a potent serum causes blanching over an area of an inch or more in diameter in cases of scarlet fever and this test is sometimes of value in differential diagnosis. My

11. Schultz and Charlton: Ztschr. f. Kinderh. **17**:328, 1918.

practice is to give an intracutaneous injection of sterile normal horse serum at the same time and if the immune serum produces a larger area of blanching, to interpret the result as positive. If uninvolved skin is available I likewise conduct a Dick skin test, as a positive reaction along with a positive blanching reaction are acceptable as evidence of the scarlatinal nature of the rash.

This rash extinction test is also of value for estimating the curative power of antiscarlet fever serums since those giving the strongest reactions are apt to be best, although Lindsay and his colleagues state that even though a serum fails to give a positive reaction it may still be efficient in the treatment of the disease.

Abstracts from Current Literature

Anatomy and Physiology

QUANTITATIVE MEASUREMENTS OF THE EXCITABILITY OF THE CENTRAL NERVOUS SYSTEM AFTER THYROIDECTOMY AND THYMECTOMY. F. C. NEWTON, Am. J. Physiol. **71**:12 (Dec.) 1924.

Newton was led to this investigation by certain observations that suggest a decrease in nervous activity in hypothyroid organisms and an opposite condition in hyperthyroid states. Of more direct relationship and serving as the actual stimulus for the present study was the observation by Rutchi that respiratory frequency was increased much less in response to temperature elevation in thyroidectomized animals than in normal animals. Rutchi also noted that when the thymus was removed as well, this decreased excitability of the respiratory center with reference to heat became more marked and apparently permanent. Newton's own objective was the actual quantitative determination of these relations discovered by Rutchi.

Newton's apparatus consisted of an asbestos-covered box into which the experimental animals (rabbits) were placed. This asbestos chamber was electrically heated and so constructed that all external light and movement and practically all sound could be excluded. The respiratory movements were kymographically recorded by means of two rubber tambours adjusted to the subject's thorax, the animal being tied on a board within the warning chamber. Through preliminary work it was found that four minute temperature exposures gave typical and optimal results and that the most satisfactory temperature range was from 20 to 35 C. The actual experimental schedule was, therefore, formulated on this basis.

After the normal relation of respiration rate to temperature change had been determined, the animals were thyroidectomized and on recovering from the effects of the operation were retested to temperature. After thyroidectomy there was noted a definite and consistent lowering of respiratory rate in response to temperature elevation as compared to the preoperative reaction. From the same animals the thymus was now removed and after operative recovery they were studied again with reference to the respiration temperature relation. In these thymo-thyroidectomized subjects, the respiratory response to increased temperatures was diminished much more as compared with the normal response than when only the thyroids were removed. The increase in respiration rate occurred much more slowly and attained only one-half the rate for normal animals and only two-thirds that determined following thyroidectomy, as shown in the accompanying table. In response to morphine

	Average Rate of Respiration per Ten Seconds to Temperature Elevations (Adapted)															
Temperature. C.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
Normal animals	7	8	9	15	21	23	25	28	31	37	43	47	47	47	48	47
Without thyroid	6	8	10	11	14	15	18	19	21	23	27	30	31	31	32	34
Without thyroid and thymus	6	6	6	6	6	7	7	8	9	10	12	14	15	18	20	22

sulphate intravenously administered, it was found that the respiration acceleration with temperature rise was definitely diminished in the thyroidectomized animals and practically abolished after both thyroidectomy and thymectomy.

These observations lead the author to the inference, substantiating previous observations, that removal of the thyroid and thymus decreases the excitability of the central nervous system to environmental stimuli, and also that there appears to exist an "augmentary interaction" between these two glands in relation to the functional activity of the central nervous system.

THE EFFECT OF THYROID THERAPY ON THE NEURO-MUSCULAR ACTIVITY OF THE CRETIN SHEEP. H. S. LIDDELL and S. SIMPSON, Am. J. Physiol. **72**:63 (March) 1925.

Study was made by the authors of the effect of thyroxin, thyroid substance and sodium iodide on neuromuscular activity in thyroidectomized sheep which, it has commonly been observed, are subject to recurrent periods of muscular prostration. In this work three definitely cretinized sheep were utilized. One was fed 0.3 Gm. thyroid extract every other day and the second 0 5 Gm. sodium iodide; in the third 0.25 mg. thyroxin was injected subcutaneously, the same time schedule being employed as was observed with the first two animals. The effect on neuromuscular activity was estimated by means of attached pedometers which were read daily. No change was noted in the sheep fed with sodium iodide. The other two animals showed definite increase in spontaneous neuromuscular activity, most marked in the thyroxin subject but in this case manifest only after a latent period of six days, whereas in the thyroid-fed animal no latent period was observed although the actual increase was of less degree and more gradual.

RAPHAEL, Ann Arbor, Mich.

Pathology, Bacteriology and Biochemistry

PHYSIOLOGY OF THE MELTZER-LYON TEST. M. CHIRAY and I. PAVEL, Am. J. M. Sc. **172**:11-21 (July) 1926.

This is a report of experimental work on the contractility of the gallbladder, attempting to show by means of kymographic tracings that in a dog the bile "B" (gallbladder bile) obtained with the Meltzer-Lyon technic is due to contraction of the gallbladder. The authors believe that the contraction of the gallbladder and the relaxation of the sphincter of odii, following the introduction of magnesium sulphate into the duodenum, is not due to reciprocal innervation because relaxation of the sphincter of odii by means of placing of a catheter in the duodenum causes only bile from the common duct to flow into the duodenum. They believe that the introduction of magnesium sulphate and other nonspecific substances such as warm water, peptone and olive oil into the duodenum cause emptying of the gallbladder due to a reflex from the duodenum. They propose that the scope of the test is thus intended to include not only a means of investigating the patency of the common duct and the presence of infection, but also to determine the contractility of the gallbladder.

HEMORRHAGES, THEIR SIGNIFICANCE, AND METHODS OF TREATMENT. C. A. MILLS, Am. J. M. Sc. **172**:71-80 (July) 1926.

The author presents a comprehensive classification of hemorrhages, and discusses their treatment. He reports good result with the use of tissue

fibrinogen given orally. Of particular interest is his success in treating five cases of hemophilia by producing a skin reaction in these patients previously sensitized to hen or sheep serum.

PARSONS, Ann Arbor, Mich.

STUDIES IN HYPERSENSITIVENESS. XII. A STUDY OF THE ATOPIC REAGINS. A. F. COCA and E. F. GROVE, J. Immunol. **10**:445 (March) 1925.

The authors have found atopic reagins present in the serum of all subjects with hay-fever and asthma, in whom cutaneous tests with the respective atopens were positive. If skin reactivity was absent in hypersensitive subjects, as in angioneurotic edema, no circulating reagins were found. They demonstrated the circulating reagins by the injection of the serum of the sensitive persons into the skin of those nonsensitive, thereby causing a local passive sensitiveness, which could be specifically desensitized. Further studies have shown the identity of certain atopens. Atopic reagins were neutralized in vivo and in vitro, but no visible precipitate and no complement fixation were observed. This method offers a valuable means of studying atopens and reagins. Variations in dosage as well as the control of numerous other factors should disclose many facts of interest.

STUDIES IN SPECIFIC HYPERSENSITIVENESS. XIV. A STUDY OF HOUSE DUST ATOPEN IN ASTHMA. C. A. SPIVACKE and E. F. GROVE, J. Immunol. **10**:456 (March) 1925.

The serum of a patient sensitive to dust, when injected into the skin of a nonatopic subject, confers a local passive sensitivity to house dust, which can be specifically desensitized. This observation suggests that house dust is a specific excitant of atopy.

STUDIES IN SPECIFIC HYPERSENSITIVENESS. XV. ON THE NATURE OF ATOPENS OF POLLEN, HOUSE DUST, HORSE DANDER AND GREEN PEA. E. F. GROVE and A. F. COCA, J. Immunol. **10**:471 (March) 1925.

In a study comprising a few experiments, the authors find that tryptic digestion and dialysis in collodion sacs removes all of the nitrogen, but does not decrease the atopic activity of pollen and house dust extracts. However, with horse dander and pea extracts the same procedure causes a decrease in atopic activity and in nitrogen. These investigators conclude that the atopens of pollen and house dust are not protein, while those of horse dander and green pea are protein.

STUDIES IN SPECIFIC HYPERSENSITIVENESS. XVI. ON ANTIGENS. A COMPARATIVE STUDY OF THE ANTIGENIC PROPERTIES OF POLLENS, EGG WHITE AND GLUE, IN GUINEA-PIGS, AS DETERMINED BY THE INTRAVENOUS AND DALE METHODS OF TESTING IN ANAPHYLAXIS. M. WALZER and E. F. GROVE, J. Immunol. **10**:483 (March) 1925.

In using crystalline egg white as an antigen, the authors found the Dale method more precise than the intravenous. Although both methods demonstrated the tendency for guinea-pigs to become strongly sensitive or not sensitive, there

were several cases of slight degrees of sensitiveness. They found the specific sensitiveness passively transferable and mediated by demonstrable precipitins. They found that the neutralization of antibody by successive fractional additions of antigen followed the principles of Coca and Kosakae, that is, it required a definite multiple of the original quantity of antigen.

In using pollen extracts as antigen, the authors produced definite anaphylaxis with both ragweed and timothy pollen and the nondigestible, nondialyzable fractions of the same, but they found that fewer animals were sensitized and that fewer were maximally sensitized than with egg white; that the uterine contractions were slower and more irregular, and that passive transfer was very rarely produced. The pollen antigen-antibody reaction was much slower both in vivo and in vitro than in animals sensitive to egg. They were unable to demonstrate precipitins with whole pollen extracts with their usual ring technic, and they found that precipitin neutralization, as demonstrated by the Dale technic, was that of simple chemical combination. The nondigestible, nondialyzable, apparently nonprotein, fraction of the pollen extracts of both high and low ragweed were identical and almost as strong antigenically as the whole pollen extract.

Hepatic Reactions in Anaphylaxis. IX. Effects of Dehepatization on the Reactions of Certain Smooth Muscle Structures in Canine Anaphylaxis. W. H. Manwaring, V. M. Hosepian, J. R. Enright and D. F. Porter, J. Immunol. **10**:567 (May) 1925.

During anaphylactic shock in intact dogs, the authors find that the uterus, urinary bladder and intestines are thrown into sharp contraction, which, in the dehepatized dog, they are not. They offer this fact as further proof of hepatic anaphylatoxin, explosively liberated by the anaphylactic liver.

Hepatic Reactions in Anaphylaxis. X. The Hepatic Anaphylatoxin. W. H. Manwaring, V. M. Hosepian, F. I. O'Neill and H. B. Moy, J. Immunol. **10**:575 (May) 1925.

The authors find that, if the liver of an anaphylactic dog is transplanted into a normal dog, the normal dog will react to an intravenous injection of the specific protein with anaphylactic manifestations. Also that if blood after shock is collected from the liver of a dog in anaphylaxis and transferred into a normal dog, that dog will react with characteristic anaphylactic manifestations. However, blood drawn from the carotid artery two to five minutes after a dog is thrown into shock, will produce no symptoms in a normal dog. This latter observation forces the conclusion that the hepatic anaphylatoxin is not the sole factor in canine anaphylaxis.

Hepatic Reactions in Anaphylaxis. XI. Glycogen Content of the Anaphylactic Liver. F. I. O'Neill, H. B. Moy and W. H. Manwaring, J. Immunol. **10**:583 (May) 1925.

Glycogen was made to disappear quantitatively from the liver during the first fifteen minutes of canine anaphylactic shock.

THE INCIDENCE OF TWO TYPES OF GROUP II HUMAN RED BLOOD CELLS. B. S. KLINE, E. E. ECKER and A. M. YOUNG, J. Immunol. **10**:595 (May) 1925.

The authors find that there are at least two types of group II human red blood cells. The agglutinogen C of Guthrie and Huck, or X of Coca and Kline, was found to be present in 81 per cent of adult group II bloods and in 51 per cent of new-born infants.

ON THE SPECIFIC SUBSTANCES IN THE ALCOHOL EXTRACT OF THE BACILLUS OF TUBERCULOSIS. L. DIENES and E. W. SCHOENHEIT, J. Immunol. **10**:631 (May) 1925.

By using lipoid solvents, and purifying the extracts considerably, the authors find that the specific substance (using complement fixation as the criterion of specificity) may be prepared in very high potency, that it is quite different to the ordinary phosphatides and that it contains considerable carbohydrate.

THE ACTION OF PANCREATIC EXTRACTS ON COMPLEMENT. A. WORMALL, H. R. WHITEHEAD and J. GORDON, J. Immunol. **10**:587 (May) 1925.

The authors find that pancreatin extract acting on guinea-pig serum first destroys the albumin fraction and then the globulin fraction, so that the complete destruction of complement action is brought about, as with heating to 56 C.

AGGLUTININ ABSORPTION. C. KRUMWEIDE, S. COOPER and D. J. PROVOST, J. Immunol. **10**:55 (Jan.) 1925.

In this much needed critical study of agglutinin absorption, Krumweide and his co-workers demonstrate the necessity for purity of the reagents, both antiserum and homologous culture. They point out the inadequacy of the absorption of common agglutinins as a criterion of likeness, and insist that the absorption of specific agglutinins must be used to determine agglutinative identity. This indicates that the antibody stimulating property of a bacterium is the fundamental one, and that agglutination and agglutinin absorption are only measures of this function. It implies that a bacterium is capable of absorbing all the agglutinins stimulated by it. This the authors hold to be fundamentally valid, although certain exceptions occur with degraded variants. Information of any value is obtained from the absorption of polyvalent serum only if the cultures used for immunization are available, and if all the cultures are tested after each absorption. In discussing the "double receptor analysis" method, they find that it has no advantage over the regular absorption method when applied simply as a means of identification or differentiation, and although they admit that suggestive data as regards types of clumping, etc., are disclosed, they condemn the use of an hypothesis, which makes an unwarranted assumption of knowledge as to what actually underlies serologic specificity. They discuss colony variants in the typhosus-paratyphosus group, the specific and group components and degraded variants. They substantiate their skepticism concerning numerous reports of variation in the infected host or from host to host. Finally, in view of their proof that there is no other means of measuring the specific agglutinin content of a serum except by using the

bacterial type which is known to have stimulated the agglutinins, they condemn any attempt to identify an infecting organism by the absorption of a patient's serum with several possible types.

GUY, Peking.

THE FUNCTIONS OF THE REGION OF THE INFUNDIBULUM AND THE TUBER CINEREUM AND THEIR RELATIONS TO THE HYPOPHYSIS. GUSTAVE ROUSSY, Ann. de méd. **18**:407, 1925.

Many physiologic functions which were attributed to the internal secretion of the hypophysis are now considered as being dependent on centers surrounding the third ventricle. In his experiments on 149 dogs and forty-six cats, Roussy has checked up earlier publications and has found new facts, which support the following conception of the rôle of the vegetative centers of the region of the tuber cinerum: The paraventricular nuclei contain a center for the regulation of the water metabolism; their artificial lesion produces polyuria and transient glycosuria. Other tuber nuclei regulate the fat metabolism; their lesion is followed by Froehlich's syndromes which could not be produced merely by removing the hypophysis without a lesion of the tuber cinereum. The only function now attributed to the hypophysis (its anterior lobe) is the regulation of the growth of the skeleton, which could not be influenced by artificial lesions of the tuber region.

THE FUNCTIONAL RELATIONSHIP BETWEEN THE PITUITARY BODY AND THE TUBER CENTERS. RÉMY COLLIN, Ann. de méd. **18**:428, 1925.

The connection between the hypophysis and the tuber cinereum is not only nervous — myelinated fibers running within the infundibulum — but also hormonic. Collin has shown that the colloid of the middle lobe of the hypophysis can also be demonstrated in the infundibulum and in the perivascular spaces of the tuber cinereum. He emphasizes that the vegetative centers of this region are under the regulating influence of the hypophysis hormone and do not act as independently as recent publications suggest. In his opinion, the overestimation of these vegetative centers without taking into consideration their correlation with the hypophysis will prove as one-sided as was the former endocrinologic point of view.

WEIL, New York.

CLINICAL INVESTIGATIONS USING THE SPEED OF SEDIMENTATION OF BLOOD CELLS AS AN INDICATION OF PROGNOSIS. OLE BOUET, Ugesk. f. Læger. **10**:240, 1926.

In thirty-five cases, mostly of rheumatic fever, the sedimentation test was used as a check on prognosis and dosage of foreign protein.

The author thinks the sedimentation test is, in many instances, more diagnostic of bodily changes than temperature fluctuations, in that it registers objective changes in the blood.

INVESTIGATION OF THE PROPERTIES OF AN INSULIN-COMPLEMENT. C. LUNDSGAARD, S. A. HOLBØLL and A. GOTTSCHALK, Ugesk. f. Læger. **11**:273, 1926.

This insulin-complement is a substance or principle which is present in the muscle tissue of cold-blooded and warm-blooded animals. It is destroyed in two minutes at a temperature of 70 C., and cannot be separated from the intact cell structure.

INVESTIGATION OF THE DIASTATIC VALUES IN THE URINE. J. FABRICIUS-MØLLER, Ugesk. f. Læger. **12**:287, 1926.

The author thinks a determination of the diastatic ferment should be carried out on all subjects complaining of acute abdominal pain. The determination is easy and reliable, and normal reading according to Wohlgemuth's scale lies between 64 and 128. In patients suffering from acute pancreatitis, the reading may be increased several hundred to several thousand. The extent of the involvement is indicated by the increase in reading. Ether anesthesia and inflammation of the salivary gland also give an increased reading.

INVESTIGATION OF THE SPEED OF GLYCOLYSIS IN HUMAN BLOOD UNDER DIFFERENT CONDITIONS. S. A. HOLBØLL, Ugesk. f. Læger. **22**:547, 1926.

The glycolytic process in human blood takes place best at body temperature, and is not influenced by the slight alkaline change which takes place in drawn blood.

Fluorine salts inhibit completely the glycolytic enzyme, while oxalate strongly retards; but in citrated, hirudinized and defibrinated bloods the glycolysis is not affected. This glycolytic enzyme is found chiefly in the red blood corpuscles.

SCHLUTZ, Minneapolis.

CONTRIBUTIONS TO THE INFORMATION CONCERNING LIVER FUNCTION TESTS. PART 3. ON THE COLOR DIAGNOSTIC SIGNS FOR THE LIVER. TŌRU OKA, Tohoku J. Exper. Med. **7**:94, 1926.

Determinations of the relative amounts of dyes in the bile and urine of dogs after intravenous injections of 0.1 cc. of 5 per cent solutions were made with twenty-nine dyes. Correlation of these results with studies of certain physicochemical properties (acid-alkaline reaction, kataphoresis, Tyndall's phenomenon and collodion membrane permeability) of the dyes indicates that those in high grade colloidal states and those in semicolloidal solutions (nearly true solutions) whose membrane permeability is impaired by being mixed with serum both eliminated through the liver, while the semicolloids which do not lose membrane permeability when mixed with serum are excreted through the kidneys.

STUDIES ON GAS METABOLISM AND MINUTE VOLUME. PART 4. GAS EXCHANGE AND MINUTE VOLUME IN ICTERUS. TSUTOMU ODAIRA, Tohoku J. Exper. Med. **7**:106, 1926.

In obstructive jaundice following the ligation of the ductus choledochus, the oxygen requirement and minute volume decrease were not in proportion to the increased icterus. From 0.5 to 1.0 mg. of atropine-sulphate given intravenously raised the gas exchange and minute volume practically to normal, doubtless by removing the parasympathetic nervous control which may be as important as the destruction of the liver function in effecting the changes in gas metabolism. Injections of epinephrine had more effect on the icteric rabbit than on the normal animal, so the sympathetic system may also be affected. Neither drug influenced the bilirubin increase in the blood.

ETHEL B. PERRY.

Nutrition

BREAST MILK CONCENTRATED BY DEHYDRATION. J. R. THOMPSON, Arch. Pediat. **43**:303, 1926.

A technic is described by means of which breast milk is concentrated by boiling to half of its original volume, and so fed to infants who will not take enough of the original breast milk to gain satisfactorily. In premature or very weak infants, this method finds its chief application. Six case reports are given to show the increase in weight made under this method.

THE DIFFERENCES BETWEEN COW'S MILK AND WOMAN'S MILK. VICTOR E. LEVINE, Arch. Pediat. **43**:353, 1926.

A very complete presentation of the constituents of cow's milk and breast milk, with tables giving analytical figures, may be found in the complete article. An unusually complete bibliography is appended.

THE RELATIVE EFFICIENCY OF CERTIFIED AND PASTEURIZED MILK IN INFANT FEEDING. M. LADD, H. W. EVARTS and L. W. FRANKS, Arch. Pediat. **43**:380, 1926.

The use of certified milk (raw) without orange juice or cod liver oil gave a considerably greater percentage of weight development than either pasteurized milk alone or such with orange juice and cod liver oil. Its protective properties against rachitic changes, as shown by roentgenograms, were clearly demonstrated and dentition developed normally. It is quite possible that the greater efficiency of certified milk over pasteurized milk is due to the more exact and scientific feeding of the cows in the particular supply of certified milk used and not to the quality of rawness. The use of orange juice and cod liver oil as a routine in all babies over 3 months of age, however fed, is well justified.

GENERAL PRINCIPLES GOVERNING MY INFANT FEEDING PROBLEM. CHARLES G. KERLEY, Arch. Pediat. **43**:478, 1926.

Well infants, regardless of age, present few feeding difficulties. Fresh cow's milk supplies a satisfactory breast milk substitute. The fresh cow's milk should be cooked for reasons of possible contamination, and when cooked with starch is rendered more digestible. Infants with severe digestive derangement do admirably on evaporated milk. It is as flexible as fresh milk and lends itself to necessary manipulation. Cooking evaporated milk with starch makes it easier of assimilation. In using cooked milk, orange juice should always be given. The administration of cod liver oil is a wise procedure in most bottle fed babies

VON MEYSENBUG, New Orleans.

INSULIN IN INFANT FEEDING. HYMAN GREEN and GEORGE ROBBINS, Boston M. & S. J. **194**:1162 (June 24) 1926.

Nine cases, considered the worst in the wards, were selected because of past history of failure to gain and of being difficult feeders. The dose of insulin was based on the ratio of 1 unit of insulin per 15 Gm. of carbohydrate in most of the cases. All patients gained except one with a serious congenital heart lesion. In six, the gains were striking. Improvement in the general condition was seen in five patients. One patient had a doubtful reaction.

GENGENBACH, Denver.

RICKETS: ITS ETIOLOGY, PATHOGENESIS AND TREATMENT. A. GRAEME MITCHELL, Internat. Clin., Series 35, **4**:117, 1925.

The three important factors which contribute to the development of rickets, are lack of sunlight, the proportions of calcium and phosphorus in the diet, and certain elements having to do with the calcification of bone, present in cod liver oil and other substances. Geography and climate, due chiefly to their influence on amount of available actinic rays of sunlight, determine to considerable extent the distribution of the disease.

In human beings, diet alone probably does not produce rickets independent of other factors. In experimental animals, rickets can be produced by feeding either a high or a low calcium or phosphorus; in other words, by a disturbed ratio between the proportions of these two elements in the diet. Ultimately, however, rickets is probably due to some as yet unknown factor which has to do with the retention of these substances. Starvation favors the deposition of calcium in the bones, while overfeeding with rapid growth has the opposite effect.

In the prophylaxis of rickets, the author feels that heliotherapy is of more importance than cod liver oil. However, during the winter in the temperate zone, the latter may be administered almost as a routine. Overfeeding with amylaceous foods and condensed milk should be avoided, and breast milk used when possible. Green vegetables and egg yolk should be added to the diet as soon as possible, and care should be exercised not to overfeed the child with cow's milk during the second year.

Once the condition has developed, the fact must not be lost sight of that, directly or indirectly, rickets involves not only the bones, but the gastro-intestinal tract and the respiratory and nervous systems as well, and that consideration must be given to these if effective treatment is to be instituted. With proper diet, exposure to sunlight and the administration of antirachitic substances, rickets of a degree sufficient to cause clinical symptoms can be prevented.

SMITH, Ogden, Utah.

JAMES LIND AND SCURVY. RALPH STOCKMAN, Edinburgh M. J. **33**:329 (June) 1926.

The writer very properly laments the fact that this eighteenth century physician of the British navy has been neglected and not given the credit due him. James Lind studied the great ravages of scurvy, and to him is due the credit for the disappearance of this disease on shipboard. He wrote a "Treatise of the Scurvy" while in practice in Edinburgh, a book which passed through three editions. While he was not the first to know something of the nature and influence of diet in scurvy, he recognized the specific value of fresh fruits and vegetables, and proposed that concentrated lemon and orange juice be prepared and used on voyages. Although outbreaks of scurvy continued to develop on the high seas, it was through no fault of Lind, as he published the information which he had gained. Lind had the faculty of scientific investigation. Other services which he rendered to civilization of his day were the interesting observations on the prophylaxis of malaria and the discovery of a method for distilling sea water for use on ship board.

No outstanding work followed his communications for over a hundred years, for it was late in the nineteenth century that Cheadle and Barlow published their descriptions of infantile scurvy, emphasizing the etiologic relation of diet.

NEFF, Kansas City, Mo.

INSULIN IN CHRONIC NUTRITIONAL DISTURBANCES OF INFANTS. O. TEZNER and A. EBEL, Med. Klin. **12**:56, 1926.

In healthy infants no water retention was seen after injection of insulin. Seven infants with chronic nutritional disturbances were treated with insulin. (Dosage: 2 by 5 to 3 by 7 units per day.) Effect on the weight could be seen only in one instance. No increase of the appetite was noted.

ZENTAY, St. Louis.

PROTEIN BODY THERAPY AND ITS ACTION ON CHILDREN AND ON ANIMALS. A. F. TUR, Ztschr. f. Kinderh. **40**:322, 1925.

Tur, in Masslow's Clinic in Leningrad, using cow's milk, human milk, and horse serum, found that protein body therapy had, out of twenty-one children ill with various diseases, a good effect in only a very small number, and that it had its best effect in hypotrophic babies. When it acted well, it acted quickly after one or two injections. In cases reacting positively, some ferments, like catalase, lipase and pepsin, were increased in strength and to this fact is due, perhaps, the good effect. In the successful cases, there was an increased activity of the blood forming organs. There was no outspoken influence of repeated injections of milk on the activity of ferments in healthy rabbits.

PERIODIC VARIATIONS OF THE ENERGY OF FERMENTS IN THE URINE OF BABIES, ONE YEAR OLD, HEALTHY, OR ILL WITH DIFFERENT DISTURBANCES OF NUTRITION; THE RELATION OF THESE VARIATIONS TO DIFFERENT PHASES OF DIGESTION. A. F. TUR, Ztschr. f. Kinderh. **40**:331, 1925.

There are two types of variations in the amount of pepsin and of amylase in the urine, which run parallel to these ferments in the blood, in relation to the stages of digestion. 1. There is in healthy babies a decrease at the height of digestion, and an increase at the end of digestion, in the stomach and in the duodenum. The picture is especially sharp for pepsin, less so for amylase. 2. In athreptic babies there is a decrease during digestion, and no increase three hours afterward. Premature babies belong to this class. Amylase and pepsin were found in the urine of the smallest breast fed babies. The energy of the ferments is less in babies the first year than it is in older children, but it reaches the normal at 10 to 12 years.

PATHOLOGIC CHANGES IN THE STRUCTURE OF LEUKOCYTES IN THE ACUTE DIARRHEAS OF INFANTS. F. HEISSEN, Ztschr. f. Kinderh. **40**:360, 1925.

Heissen, from a study of twelve cases of intoxication, and forty-two cases of dyspepsia, reports that abnormal granules and nuclei and early forms of polymorphonuclear neutrophils regularly occur in the leukocytic picture of cases of acute diarrheas in babies, especially of intoxication, to a degree seldom found in any other disease. Diagnostically, this is of no value, but from the point of view of prognosis, it is, for the greatest changes occur in the most severe cases. The presence of eosinophils and of monocytes, which regularly disappear in intoxication, is of good import. The white count of subtoxic dyspepsia (cases with high fever and disturbances of consciousness without deep breathing) and of mild diarrheas is within normal limits.

EMERSON, Boston.

HYPERACIDITY IN INFANTS. G. TOROK, Orvosi hetil. **69**:789, 1925.

Hyperacidity in infants is only found if it is the result of a general disturbance of metabolism. In exudative diathesis it is due to a disturbance of the chlorine metabolism, in new-born infants it is due to congenital alkalinity, in dyspepsia to acquired hypoalkalinity. In intoxication combined with acidosis, it is due to the acidosis. Neurosis as a primary factor can be entirely neglected.

ZENTAY, St. Louis.

Prenatal Conditions

THE DEVELOPMENT OF PRENATAL CARE IN DETROIT. WALTER E. WELZ, Am. J. Obst. & Gynec. **11**:671, 1926.

The report covers the results obtained in Detroit's Municipal Prenatal Clinics for the years 1922 to 1924.

The maternal mortality, which was largely due to sepsis, has been cut in half as compared to all the other cases in the city. This fact has lowered the morbidity probably more than all of the other factors together.

They obtained a reduction of sixteen stillbirths per 1,000 living births as a result of prenatal care. This reduction was particularly true among the colored patients. There was a reduction of seventeen neonatal deaths per 1,000 among white infants and thirty-eight in colored. A large number, 30 per cent of all the cases, were treated for some intercurrent disease during their pregnancy. Fifty per cent of the colored women were vaccinated, and a routine adopted of vaccinating all new subjects who had not had a successful vaccination in five years.

Fourteen per cent of the cases were syphilitic, and received prompt and vigorous treatment. Clinical results obtained indicate that if adequate maternal treatment was instituted before the first sixteen weeks of pregnancy, transmission to the fetus was prevented. The average for the marked toxemias of late pregnancy was 3 to 4 per cent, and the fetal mortality was 200 in each 1,000 cases. There was an infant mortality of 8 per cent in the 6.6 per cent of maternal heart cases.

None of the tuberculous subjects had induced labor. There was an infant mortality of 77 in 1,000 deliveries. By conserving the maternal vitality throughout pregnancy, a material decrease in infant mortality resulted and a number of the mothers improved in health in spite of their infection.

No permanent morbidity resulted from delivery in the cases of contracted pelves, and only four primary fetal deaths in 136 cases.

Sixty per cent of the neonatal deaths resulted from prematurity.

About 4 per cent of all pregnant women in Detroit are cared for by the Social Service Clinic, banded together in protecting and promoting the health of the mother and child.

ALLEN, Chicago.

A RARE CASE OF MULTIPLE CONGENITAL ATRESIA OF THE ILEUM WITH COMPLETE SEPARATION OF A SEGMENT OF BOWEL. I. H. ERB, Am. J. Path. **2**:137, 1926.

An interesting case of multiple atresia of the ileum with complete separation of a segment of bowel is here reported. Such an isolated segment is, so far as we have been able to determine, peculiar to this case. The absence of bile pig-

ment below the upper atresia would indicate that the occlusions had occurred before the third or fourth month of intra-uterine life. The presence of a peritoneal adhesion suggests that the cause of these atresias was an early intra-uterine peritonitis.

AUTHOR'S SUMMARY.

FAMILIAL FINGER CONTRACTURE AND ASSOCIATED FAMILIAL KNEE-JOINT SUBLUXATION. DOUGLAS P. MURPHY, J. A. M. A. **86**:395 (Feb. 6) 1926.

The pedigree of a family presenting congenital finger contractures and associated subluxation of the knee-joint is given. About one third of the members were so affected.

CONGENITAL ABSENCE OF THE ESOPHAGUS. ROBERT E. PARRISH, J. A. M. A. **86**:33 (Jan. 2) 1926.

At necropsy, an atresia of the esophagus extended from the cricord cartilage to the cardia, where a small pouch projected up from the stomach to the diaphragm. The atresia was complete. No other congenital abnormalities were noted.

CONGENITAL ATRESIA OF ESOPHAGUS ASSOCIATED WITH ATRESIA OF ANUS TRACHEA; ESOPHAGEAL FISTULA. MAX THOREK, J. A. M. A. **86**:33 (Jan. 2) 1926.

The atresia occurred at the middle third of the esophagus, and was associated with an esophageal fistula, atresia of the trachea and of the lower portion of the rectum and anus.

BONAR, Salt Lake City.

MACROSOMIA OR CONGENITAL HYPERTROPHY. E. D. FENNER, M. J. & Rec. **123**:501 (April 21) 1926.

Macrosomia or gigantism has been defined as "A monstrous largeness of the body." Fenner reported a case in a girl, aged 7 years. The patient was admitted to hospital in 1921, on account of a swelling over the left tibia which followed a blow and which was increasing rapidly in size. The family and the past history of the patient were negative, except for an attack of grip during the epidemic of 1918. The enlargement of the leg was noticed at birth and it had become more noticeable as the child advanced in age. On admission, the left lower limb was about 5 inches longer than the right and about twice as large. The muscles were flabby and the skin was rough and coarse. The left labium majus was markedly hypertrophied. There was a large soft swelling attached to the bone over lower half of left tibia. It was not painful or tender.

Skiagraphs showed gigantism of the entire skeleton of the lower left extremity, coxa valga and shadow of the tumor. An exploratory incision over the tumor was made, and a piece sent for examination was reported to be fibrosarcoma. A transcondyloid amputation through the knee joint was performed. Healing was delayed by an obstinate watery discharge from the stump, but ultimately the patient was discharged with the wound healed.

WILLIAMSON, New Orleans.

Diseases of the New-Born

THE MECHANICS OF BIRTH INJURIES, THEIR CAUSE AND PREVENTION. J. W. NEUMANN and WALTER E. LEVY, Am. J. Obst. & Gynec. **11**:645, 1926.

Recent statistics concerning the lowering of fetal mortality are misleading. More careful prenatal care is probably the largest factor, while the birth injury element still keeps up its high percentage.

Pediatricians and neurologists should combine their demands with those of the expectant mother that these injuries be decreased to the minimum by accurate, careful, specialized obstetric care. The authors feel that 75 per cent of these injuries can be eliminated in expert hands by adherence to the following rules:

1. Absolutely avoiding the use of high forceps.
2. Not applying forceps to the after-coming head in breech deliveries.
3. Noninterference, except for very definite maternal and fetal indications. More judicious use of pituitary extract.
4. More care in resuscitation, avoiding use of sponges to clear the throat or too early mouth to mouth insufflation.
5. The more thorough training of attendants at childbirth.

CISTERN PUNCTURE IN INTRACRANIAL BIRTH INJURIES. BERMAN S. DUNHAM, Am. J. Obst. & Gynec. **11**:833, 1926.

The author reports 78 punctures of the cisterna magna for diagnostic and therapeutic purposes. He did not observe any resulting harm in any subject, although one infant, under treatment for cerebrospinal fever, had 26 punctures. He has varied the usual technic by inserting the needle a few millimeters above the posterior rim of the foramen magnum, and then displacing the soft tissues downward, slips the needle under the posterior rim of the occipital bone. It is then pushed upward and forward until the "give" is felt, indicating piercing of the occipitatlantal ligament.

Ten new-born infants, nine of whom had intracranial hemorrhage, received 27 cistern punctures. The general condition of the babies when first seen were: One case, poor; seven cases, critical, and two cases moribund. Preliminary lumbar puncture was done in five cases, with three dry taps. Six patients recovered and are now normal.

Two of the four deaths occurred in premature infants; another death occurred in a child with delayed coagulation who was moribund. The other infant was moribund when first seen.

PURPURA HEMORRHAGICA COMPLICATING PREGNANCY. REPORT OF A CASE IN WHICH BOTH MOTHER AND CHILD WERE AFFECTED AND RECOVERED. PHILIP LIEBLING, Am. J. Obst. & Gynec. **11**:847, 1926.

Purpura hemorrhagica is a very serious, but fortunately a rare condition complicating pregnancy. The case report brings the total cases reported up to forty-eight. Only eight of these occurred in both mother and child.

The petechia in this case began in the fifth month of pregnancy. They appeared in successive crops, accompanied by mild epistaxis and bleeding from the gums. The blood count at this time was: erythrocytes, 2,400,000; hemoglobin, 45 per cent, and blood platelets 40,000. The mother was treated with calcium lactate and saccharated ferrous carbonate, 20 grains each three times a day.

The condition gradually improved, the platelet count going up to 200,000 and the hemoglobin to 77 per cent.

The patient went into labor at term. The baby weighed 8 pounds and 11 ounces, and had several petechial spots on the face, trunk and extremities. At different times in the next few days the vomitus, urine and stool contained traces of blood. Five cubic centimeters of thromboplastin was injected into the buttock. The blood count at this time was: erythrocytes, 6,000,000; hemoglobin, 110 per cent, and platelets 40,000. The petechia on the baby gradually faded and did not reappear. The mother, however, had another crop about two weeks later. These disappeared soon. The patients remained under observation for about one month, and both remained free during this period.

ALLEN, Chicago.

SCLEREMA NEONATORUM. G. A. HARRISON, Arch. Dis. Child. **1**:123 (June) 1926.

The study of sclerema is concluded with a chemical investigation of the subcutaneous fat. The melting points, iodine numbers, cholesterol content, acid values, and neutralization values are determined in four cases. Normal controls were studied for comparison. The eighteen cases of sclerema with chemical investigations reported in the literature are reviewed and the theories of causation discussed. In the five cases studied the following characteristics were found:

1. The fat was of solid consistency.
2. The histologic picture was one of chronic inflammation with giant cell formation and calcification.
3. Anisotropic crystals are seen in frozen sections.
4. Roentgenograms show calcified nodules.
5. There is an excess of calcium and phosphorus in the subcutaneous fat, also an excess of cholesterol.
6. The separated fat and ether extracts have a raised melting point.
7. The iodine number and saponification value of the subcutaneous fat are within normal limits.

PETERMAN, Milwaukee.

INTRACRANIAL HEMORRHAGE OF THE NEW-BORN. JOHN A. FOOTE, Internat. Clin. Thirty-Fifth Series **4**:130 (Dec.) 1925.

Two varieties may be recognized: A fulminating type, which if not rapidly fatal may lead to Little's disease, and a slow type which may also prove fatal, but which is more amenable to treatment. The latter form is especially discussed in this paper. Prematurity, microcephaly and abnormally rigid skull are possible predisposing causes.

Symptoms may appear within the first few days postpartum, or may not become manifest for three or four weeks. In the latter case, they seem most likely to follow violent or continuous crying. Refusal of the breast, after having nursed normally for some days, is an early and significant sign. Twitchings or tonic spasms from increased cerebral pressure, cyanosis from pressure in the region of the medulla and head retraction from basilar hemorrhage, may occur. The fontanels may not be tense in subtentorial hemorrhage. A peculiar protrusion and retraction of the tongue (Foote's sign) may be noted. In some cases the clotting time of the blood is prolonged, and there may be bleeding into the skin and mucous membranes. The spinal fluid contains bright red or clotted blood when the hemorrhage is below the tentorium. Should the

infant survive, a type of Little's disease may develop if the motor areas have been involved, but more commonly there develops a cerebellar palsy from bleeding below the tentorium.

Treatment consists in absolute rest. The infant should not be disturbed for bathing, and should not be put to the breast for one or two weeks, being carefully fed in the meantime by gavage or feeder. The author routinely gives an intraperitoneal transfusion of citrated blood. Pressure should be relieved by lumbar or cisterna puncture, repeated if necessary.

SMITH, Ogden, Utah.

THE EFFECT OF GAS ANESTHETICS USED IN LABOR. N. SANFORD HEYWORTH, J. A. M. A. **86**:265 (Jan. 23) 1926.

The blood of 600 infants was examined at birth and during the first ten days of life. It was found that nitrous oxide gas administered during labor caused a prolongation of the average bleeding time at birth of one minute, and the coagulation time was lengthened two minutes. Both corresponded to the normal in nine days. Ethylene was found to prolong the bleeding two minutes and the coagulation time three minutes. In nine days, however, they became practically normal.

BONAR, Salt Lake City.

PERFORATION OF STOMACH IN INFANCY. J. F. BRINKMAN, J. Iowa M. Soc. **16**:293 (June 20) 1926.

Brinkman reports the case of a twin, weighing 5½ pounds, whose family history was negative. The child developed peritonitis on the third day, vomited and no peristaltic action was noted. Distention increased.

An exploratory examination showed blood-tinged fluid in the peritoneal cavity, with diffuse inflammation of the visceral peritoneum. Inspection disclosed gas bubbles escaping from the stomach at about the middle third of the greater curvature. The opening was closed, and the child lived about ten hours after operation.

TURNER, Des Moines, Ia.

SIGNS OF ACUTE PROVED CASES OF INTRACRANIAL HEMORRHAGE IN THE NEW-BORN. A. S. MACLAIRE, M. J. & Rec. **123**:215 (Feb.) 1926.

The cause of intracranial hemorrhage of the new-born can be chiefly attributed to one of the following groups: (1) Trauma; (2) asphyxia; (3) hemorrhagic disease; (4) syphilis of the new-born.

Nine per cent in a series of 500 consecutive new-born babies, on whom a routine diagnostic lumbar puncture was performed within forty-eight hours after birth contained blood or blood elements in the spinal fluid. The author then gives a short résumé of the types of labor which occurred in this particular series.

The most frequently observed sign in this series was twitching of the hand. Other signs were twitching of the feet, cyanosis, poor nursing, convulsions, respiratory difficulties, a weak cry or failure to cry, and frothing at the mouth. Treatment consists of repeated lumbar puncture every six to twenty-four hours until clear fluid is obtained. If after three or four days the cerebrospinal fluid remains unaltered, cranial drainage must be instituted.

Improperly treated cases result in: (1) Varying forms of spastic paralysis with or without mental impairment; (2) hydrocephalus of the communicating or complete internal type.

WILLIAMSON, New Orleans.

SOFTNESS OF DOME OF SKULL IN THE NEW-BORN. HANS ABELS, Wien. klin. Wchnschr. **39**:653 (June) 1926.

The localized form is described, in which there is abnormal softness due to defective ossification, usually affecting the median borders of the parietal bones. The most frequent form is the "sagittal fontanel," a small triangular or round soft spot on the sagittal suture above the small fontanel. The area of softening may be more extensive. The localized type is to be differentiated from generalized softness of the skull, although it has some of the characteristics of mild grades of the latter—abnormally large fontanels, gaping sutures and yielding bone edges. Both varieties are not to be confused with rachitic softening of the skull, which is never manifested at the time of birth.

On the basis of clinical observation of about 4,000 new-born infants, Abels concludes that a constitutional deficiency of osteogenesis is involved in local as well as in general softening. The infants examined usually showed other signs of retarded development. Cases were more frequent in winter months.

In localized softness of the dome of the skull, of which there were 338 cases in his series, the author believes that there is an additional factor involved in the pathogenesis; this is, the mechanical pressure of the fetal skull against the maternal bony pelvis. A resultant interference with osteogenesis occurs at the points of pressure, especially that portion of the skull which impinges on the linea innominatis. Abels offers in support of his theory the observation that the condition was never found in infants born in breech presentation. The location of the softness varied according to the obstetric position of vertex cases. In primiparas, the new-born infants were more frequently affected than the infants of multiparas, probably due to the less relaxed uterine musculature forcing the fetal skull against the bony pelvis of the mother.

OLMSTED, Seattle.

TREATMENT OF EXTREMELY WEAK PREMATURE INFANTS WITH BLOOD SERUM OF PREGNANT WOMEN. R. RAISZ, Orvosi hetil. **69**:1061, 1925.

Blood serum of pregnant women is used in every premature infant weighing less than 2,000 Gm., and in others if they do not gain under the usual forms of treatment. Two hundred and forty-seven premature infants were observed. The total mortality is twenty-seven (10.9 per cent), but the mortality of the 196 not treated with blood serum is twenty-four (12.2 per cent), while of those treated with serum only three died (5.8 per cent).

The serum was used in small doses of 1 cc. Injections were given subcutaneously and at more or less frequent intervals.

ZENTAY, St. Louis.

Acute Infections

CHARACTERISTIC CHANGES IN BLOOD CHEMISTRY IN WHOOPING COUGH. JOSEPH C. REGAN and ALEXANDER V. TOLSTOONHOV, J. A. M. A. **86**:181 (Jan. 16) 1926.

A total of 200 analyses of the blood were made. Distinct and apparently significant changes have been encountered, the most characteristic of which were a lowering of the hydrogen ion concentration and a diminution of the inorganic phosphorus content of the plasma.

BONAR, Salt Lake City.

A Scarlatinal Infection Due to Streptococcus Viridans. Edward Steinfield, J. A. M. A. **87**:241 (July 24) 1926.

The acute infection to which Steinfield calls attention was prevalent during the winter of 1925-1926. The onset was sudden, with fever from 101 to 103 F. The throat was markedly injected. A scarlatinoid exanthem appeared in from twenty-four to forty-eight hours. Hemorrhagic nephritis frequently occurred at the same time. The duration of the febrile period was from one to two weeks. The condition was differentiated from scarlet fever by its infectivity for all ages, the atypical character of the exanthem (small papular as well as punctate rash) and the subsequent infection with true scarlet fever of two cases. Cultures from the throat and exudates elicited *Streptococcus viridans* in contrast to the hemolytic streptococcus found in scarlet fever.

[Arch. Dermat. & Syph.]

Complexity of the Scarlet Fever Toxin and Antitoxin. A Preliminary Report. W. H. Par and R. G. Spiegel, J. Immunol. **10**:829 (Sept.) 1925.

The authors find that an individual convalescent serum neutralizes the toxin of a certain scarlet fever streptococcus in certain susceptible persons and not in others. They therefore conclude that the toxic filtrate contains more than one toxic substance; one filtrate might contain toxin A, B, C and D; another A, C, D and E; another B, C, D and E, etc. Antitoxins would be developed for each specific toxin, although in different quantities.

Guy, Peking.

Measles Complicated by Gangrene of the Legs. D. I. Hishon, Proc. Roy. Soc. Med. (Section for the Study of Disease in Children) **19**:35 (March) 1926.

The patient, an infant, aged 16 months, was admitted to hospital on Oct. 23, 1925, on the second day of the rash of measles with signs of commencing pneumonia. The general condition was poor. From October 28 to November 3, the patient became progressively weaker. The legs and feet were cold and blue. On November 3, patches of discoloration were noticed on the dorsal surface of the feet, the soles were cold and blue. The femoral and popliteal pulses could not be felt. By November 5, the line of demarcation was obvious on both feet. On November 8, the patient died. Necropsy disclosed that the femoral popliteal arteries contained antemortem bloodclot and the left kidney showed a pale infarct. The liver and spleen were engorged.

Williamson, New Orleans.

Pneumococcus Meningitis Treated with Morgenroth's Optochin Hydrochloride. Report of a Case with Recovery. H. L. Ratnoff and A. M. Litvak, Arch. Pediat. **43**:466, 1926.

Many authors are quoted as expressing the conviction that pneumococcus meningitis is a very fatal disease, with a mortality of over 95 per cent. The case reported is that of a girl, aged 7½ years, who complained of pain in the neck, stiffness of the back, and fever, coming on suddenly. No pathologic change was found except in the spinal fluid, which was at first clear but later became purulent, and which showed up to 2,000 cells, all polymorphonuclears. Smear and culture showed pneumococci present. Under intraspinous antipneumococcus serum therapy the symptoms became worse and the child showed no subjective

improvement. Fifteen cubic centimeters of 1:1,000 solution of ethyl-hydrocuprein in sterile saline solution was given intraspinously twice a day for three successive days and then once a day for three more days. The organisms disappeared and the fluid became much clearer, while the patient became much improved. As blurred vision was complained of, the ethyl-hydrocuprein was discontinued and serum alone was given in amounts of 20 cc. intraspinously. The condition became rapidly worse, pneumococci reappeared in the spinal fluid. Ethyl-hydrocuprein was given again every third day, 15 cc. of 1:1,000 solution in saline solution. This brought about complete recovery of the patient.

It is pointed out that the use of serum and ethyl-hydrocuprein together enhance the value of both, and that the doses used of each in this case were so small that any one, used alone, would have had no therapeutic effect. Hence both antipneumococcus serum and ethyl-hydrocuprein should be used.

ACUTE TYPHOID CHOLECYSTITIS IN CHILDREN. REPORT OF A CASE TREATED WITH MERCUROCHROME. MENDEL ZELIGS, Arch. Pediat. **43**:485, 1926.

A case of acute typhoid cholecystitis in a child, aged 12 years, is reported. The pain, tenderness and rigidity over the gallbladder were promptly relieved by nonsurgical duodenal drainage. Following one intravenous injection of mercurochrome-220 soluble, the typhoid bacilli disappeared from the bile.

VON MEYSENBUG, New Orleans.

CARONIA'S INTRADERMAL REACTION WITH THE TOXINES OF SCARLET FEVER AND OF MEASLES. A. F. HECHT, Ztschr. f. Kinderh. **40**:309, 1925.

Hecht used scarlet fever toxin supplied by Caronia, and found that in twenty-seven cases it could not be used to differentiate scarlet fever from other diseases, because cases of scarlet fever, either later in the course of the sickness or perhaps a year afterward, might show a positive reaction. Nor could any real difference be established in thirty-nine cases by the reaction to measles toxin, between those who had had, and those who had not had measles.

EMERSON, Boston.

SOME INFORMATION ABOUT THE OCCURRENCE OF PERTUSSIS AND A CONSIDERATION OF THE USE OF PERTUSSIS VACCINE. S. E. KOFOED, Ugesk. f. Læger. **24**:585, 1926.

This paper gives in detail the spread of a pertussis epidemic and vaccination of children in small villages before the epidemic had reached them; 338 children were vaccinated. Those vaccinated before incubation or during incubation had a mild attack or none. A few were very sick and had complications. A much greater number of the unvaccinated children were very sick and a large number had complications.

SCHLUTZ, Minneapolis.

Chronic Infections

THE NEWER VIEWPOINTS IN THE TREATMENT OF SYPHILIS. MOSES SCHOLTZ, Am. J. Syph. **10**:273 (April) 1926.

Scholtz points out that the recent trend in the management of syphilis is to place too much emphasis on the chemotherapy and serology, to the exclusion of the biologic factors concerned.

MCBRIDE, Iowa City.

BISMUTH SALICYLATE IN EXPERIMENTAL RABBIT SYPHILIS. O. M. GRUHZIT, Arch. Dermat. & Syph. **13**:195 (Feb.) 1926.

When administered intramuscularly to rabbits, bismuth salicylate, an insoluble compound, was found to be absorbed at about the same rate as potassium bismuth tartrate, both preparations having been administered in oil. Potassium bismuth tartrate is water soluble, though when mixed with oil and sterilized, it was found to be almost wholly insoluble. Traces of both preparations were found to be present at the site of injection as late as forty-seven days after administration. Following the administration of bismuth salicylate, bismuth was eliminated chiefly by way of the urine (from 77 to 92 per cent), the remainder being excreted in the feces. Eight to eleven injections of 0.5 to 2 mg. each per kilogram of body weight produced no kidney or liver lesions. A single dose of 5 mg. per kilogram rendered the syphilitic lesions free of spirochetes in forty-eight hours. Absolute cures were produced in all rabbits, even with the smallest dosage of 0.5 mg. per kilogram in the course of eight to eleven injections. No signs of pain or irritation were observed at the injection site.

INTRAMUSCULAR ABSORPTION OF SOME INSOLUBLE BISMUTH COMPOUNDS AS REVEALED BY THE ROENTGEN RAY. H. N. COLE, HARRY L. FARMER and HAGOPE MISKDJIAN, Arch. Dermat. & Syph. **13**:219 (Feb.) 1926.

Bismuth has been the cause of many fatalities and severe chronic poisonings. Thus the rate of absorption of a bismuth preparation becomes an important consideration in the choice of a preparation for antisyphilitic treatment. Of the various preparations studied, the two giving the most rapid absorption were potassium bismuth tartrate and bismuth salicylate, the absorption of the former being somewhat more rapid than that of the latter. Even with the tartrate, roentgen-ray shadows at the injection site were observed as late as 39 days after administration. The two preparations mentioned are preferable, therefore, to the others studied. Bismuth preparations should be administered oftener than once a week.

BLOOD CHOLESTEROL FINDINGS IN SYPHILIS AND IN OTHER SKIN DISEASES. ISADORE ROSEN and FRANCES KRASNOW, Arch. Dermat. & Syph. **13**:506 (April) 1926.

A study of the cholesterol content of the blood of patients in various stages of treated and untreated syphilis was made. They found no direct connection between the cholesterol content of the blood and the Wassermann reaction. The normal range of blood cholesterol in children is stated to be from 130 to 180 mg. per hundred cubic centimeters, almost the same as that in adults.

THE ROSE BENGAL TEST FOR LIVER FUNCTION, WITH PARTICULAR REFERENCE TO ITS USE IN THE THERAPY OF SYPHILIS. N. N. EPSTEIN and J. E. RAUSCHKOLB, Arch. Dermat. & Syph. **14**:122 (Aug.) 1926.

Arsphenamine jaundice is constantly associated with liver dysfunction. The liver function returns to normal with clinical recovery. No relation could be shown between liver dysfunction and arsphenamine dermatitis. It was thought that acute syphilitic hepatitis was detected in several instances during the secondary stage when no physical signs pointing to liver disease could be observed. The rose bengal liver function test is believed to be a valuable indicator as to the prognosis in cases of arsphenamine icterus.

JEANS, Iowa City.

REMARKS ON THE DECLINE OF MORTALITY FROM TUBERCULOSIS AMONG INFANTS AND CHILDREN. ALLEN K. KRAUSE, Boston M. & S. J. **194**:1173 (June 24) 1926.

The author first refers to observations in New York reported by Drolet. In 1898, the deaths of children constituted between one-sixth and one-seventh of all deaths from tuberculosis in New York City; in 1923 they made up less than one-tenth of the total number. Within the same period the meningitis rate (tuberculous) dropped from 353 to 51; the pulmonary rate from 84 to 22, and the peritoneal mortality from 50 to 5. J. O. Emerson showed that in Boston between 1898 and 1921, the mortality from pulmonary tuberculosis had declined the most in the 10 year age-period from birth to 10 years. The author, therefore, assumes that children's contacts with tuberculosis are being greatly reduced quantitatively, thus making for much less severe tuberculosis and death. He therefore concludes that thus, "as has happened in malaria and other infections, tuberculosis, once it has declined to a certain (as yet unknown) level or total of infectiousness, may have a way of solving itself. There may come a time when not enough tubercule bacilli 'remain at large to infect mankind sufficient to maintain a given level of mortality—*with, of course, environmental factors what they are.*' Perhaps that time is already here. If it is, the child looms larger than ever in the scheme of tuberculosis."

GENGENBACH, Denver.

GASTRIC ACIDITY IN SYPHILITIC CHILDREN. MAURICE DORNE, W. A. BRAMS and I. HARRISON TUMPEER, J. A. M. A. **86**:267 (Jan. 23) 1926.

Gastric analyses were made on twenty-one syphilitic children from 4 to 13 years of age, and on seventeen children free from syphilis or gastrointestinal disturbance. As a group, the syphilitic children manifested a hypoacidity, 9.5 free acidity and 24.9 total acidity. The free acidity of the control cases averaged 19.4 and the total acidity 41.6. With 10 years of age as the dividing line, the authors' figures demonstrated that in both age groups above and below 10 years, the free and total acidity values were markedly lower in syphilitic children than in the controls.

THE PES CAVUS OF CONGENITAL SYPHILIS. C. W. GOFF, J. A. M. A. **86**:392 (Feb. 6) 1926.

In the course of a systematic study of congenital syphilis, interesting features were brought to light, particularly in respect to pes cavus. This deformity, with its characteristic pseudohammer toes, was generally bilateral and occurred in practically every case of congenital syphilis. It did not occur in the control cases that were free from syphilis. It is apparently dependent on a neuromuscular condition for its pathogenesis. This is denoted further by a very slight scoliosis, tilting of the pelvis, muscular tautness of the hamstrings. The report of 33 cases included adults. This type of pes cavus is not progressive and rarely gives rise to symptoms. Antisyphilitic treatment seems to diminish this deformity. The author considers the finding of pes cavus a valuable aid in diagnosing obscure cases of congenital syphilis.

BONAR, Salt Lake City.

THREE CASES OF BISMUTH ABSCESS. J. GOUIN and ALLANIC, Bull. Soc. franç. de dermat. et syph. **32**:445, 1925.

Appearing from six weeks to three months after injections of bismuth hydroxide in oil, the gluteal tenderness was accompanied by malaise, nausea, vomiting and fever. The bismuth compound, and not the oil, was blamed. Incision and drainage was necessary.

VON RECKLINGHAUSEN'S DISEASE AND CONGENITAL SYPHILIS. J. MONTPELLIER, Bull. Soc. franç. de dermat. et syph. **32**:465, 1925.

A case is reported, occurring in a man, aged 25, with corneal ulcers and oral signs of congenital syphilis.

CONGENITAL SYPHILIS AND GENERAL PARALYSIS IN CHILDREN. A. MARIE, Bull. Soc. franç. de dermat. et syph. **33**:153, 1926.

Two cases of paralysis were presented, occurring in children of paretic parents. An extensive bibliography is appended.

[ARCH. DERMAT. & SYPH.]

THE PRETUBERCULAR CHILD. ARTHUR MASSEY, Brit. J. Tuberc. **20**:2 (April) 1926.

The pretubercular child is defined as one who, by reason of family history, past debilitating disease or physical condition, is likely to become a victim of the disease. Also are included those children suspected of having tuberculosis, but whose condition will not admit of a positive diagnosis.

The physical characteristics of the child with the "tuberculous diathesis" include fine, dry, delicately tinted skin, clear sclerotics and long silky lashes, high arched eyebrows, long soft hair, good teeth, long thin neck with prominent larynx, thin chest with dilated superficial veins. There may be excessive growth of hair in the midline of the back. The arms are long and thin. The interphalangeal joints of the hands are large, with hyperextension at the metacarpophalangeal articulations. These features, while typical, are seldom all present in one subject. History of repeated colds is important only when refractory, protracted cough is present.

The recognition of this type of child is of great importance, since in childhood preventive as contrasted with curative measures may be most easily and successfully applied.

RADIOGRAPHY OF THE CHEST IN TUBERCULOSIS. J. E. A. LYNHAM, Brit. J. Tuberc. **20**:2 (April) 1926.

In the pathologic sense, early tuberculosis probably cannot be demonstrated radiographically, since tissue changes must be fairly well advanced before characteristic shadows appear on the screen. Such examination is valuable in early cases, however, in that it may supply confirmatory evidence when the physical signs are doubtful. In the event of inconclusive roentgenographic and physical findings, subsequent roentgen-ray examinations may show clearing up of a doubtful focus, or, on the other hand, progressive changes which will confirm the suspicion of tuberculosis. Such examinations are especially important in children who are slow to recover from measles and whooping cough. Shadows due to nonspecific changes at the lung roots show a tendency

to clear up ultimately, while tuberculous glands are likely to persist and later show caseation. When, in addition to evidence of caseous glands, steamy opacities appear far out in the lungs, and especially when lines of increased density unite them to the root shadows, a diagnosis of tuberculosis is to be seriously considered.

Chronic heart disease, especially congenital, may produce changes in the lungs impossible of differentiation from tuberculosis.

SMITH, Ogden, Utah.

PULMONARY TUBERCULOSIS AND ALLIED CONDITIONS IN INFANCY AND CHILDHOOD. LEONARD FINDLAY, Edinburgh M. J. **33**:147 (March) 1926.

Findlay writes of his experience with pulmonary tuberculosis rather than with intrathoracic tuberculosis in a children's hospital. He regards it difficult to accept Ghon's teaching that all intrathoracic tuberculosis is pulmonary because of the impossibility of determining when the infection began. In Toronto, mediastinal tuberculosis is frequent, while in Glasgow it is rare. He considers tuberculosis as chiefly a disease of infancy and early childhood, and especially of the acute bronchopneumonic type, with generalization in the lungs.

As regards the age, the commonest incidence is in the first year, rapidly declining up to the sixth year, and thereafter relatively infrequent. His private patients also show that medical tuberculosis is the most frequent during young childhood, but in the second and third years.

Tuberculosis of the lungs in children is acute or subacute, chronic cases being rare. The outcome is fatal in a short time. Chronic pulmonary tuberculosis, so-called, not enlarged mediastinal glands, is found at necropsy to be unresolved pneumonia with bronchiectasis. Findlay states that he has not seen in a large postmortem experience any case of chronic pulmonary tuberculosis.

Pulmonary tuberculosis in children, with the exception of infants, is rarely limited to a lobe or one lung. It practically always has reached both lungs when the condition is observed. In the young infant, unilateral pulmonary tuberculosis may be found, causing consolidation from apex to base.

In the diagnosis, sputum obtained by swabbing the child's throat, the presence of enlarged glands and spleen, and in general tuberculosis, the presence of papulonecrotic tuberculids of the skin are of great help. In older children, the Pirquet reaction is of least value for it occurs in healed or latent tuberculosis as well as active.

As for the aid given by roentgenograms, the clinician should be expert in their reading. In all chests there are root shadows, some of which may be due to the length of the exposure and to the hardness of the tube. Findlay agrees with the conclusions of the United States Committee for the National Tuberculosis Association, which state that the normal chest of children varies beyond the possibility of exact description, that the hilum shadow found entirely within the inner third of the lung is normal or not conclusive unless it represents a growth of mediastinal pleurisy, and that calcified nodes at the root, when existing without lung disease, are common in normal chests and represent in some cases healed conditions, inflammatory but not necessarily tuberculous.

Acute pulmonary miliary disease does not show in the picture, but is found at necropsy to be a subacute or bronchopneumonic. Simple bronchopneumonia as well as tuberculosis may show wedge-shaped shadows reaching out from the mediastinal focus into the lung. In infants, the localization to one lung may be seen as a dense uniform shadow in the apex or one base, and if tuberculous it soon extends to the remainder of the lung and opposite side, or is followed by cavitation.

The so-called "snow-storm" picture is seen as a fine uniform mottling or a mass of nodules. If they have rounded shadows it is due to tuberculosis, but when not discrete they may be due to septic bronchopneumonia. The "snow-storm" picture is the most common and characteristic of tuberculosis.

Physical signs are slight when there is no large region of consolidation. The discrepancy between physical signs and the roentgen-ray picture furnishes a prominent characteristic of pulmonary tuberculosis. The failure to find physical signs is due to the masking of the small consolidated areas by the surrounding emphysema. General or somewhat diminished resonance with wheezing râles are the only signs that may be found, so that the roentgenogram discloses the tuberculous nature of the disease with the lungs riddled with consolidated areas. The roentgenogram is in advance of the physical signs, while in bronchopneumonia the signs are evident before the picture shows lesions.

SOME STATISTICAL ASPECTS OF TUBERCULOUS MENINGITIS. JAMES L. HOLLIDAY, Edinburgh M. J. **33**:160 (March) 1926.

The official Glasgow figures for tuberculous meningitis are discussed by Holliday, on the basis of the frequency by years, the influence of seasons, and the relationship to the conditions of living.

Tuberculous meningitis has steadily declined from 321 per million population in 1899 to 189 in 1923. The complication has not relatively changed in its incidence, but has declined along with the death rate from all forms of tuberculosis. Meningitis is an accident due to the ulceration of a gland, usually of the bronchial region, into the blood stream.

As regards the seasonal frequency of this form of meningitis, there are always twice as many cases (deaths) in May as in October. The peak of cases in the spring and early summer is evidently due to the same causes which produce a peak of respiratory infection and a tuberculous involvement of the bronchial glands. Although meningitis lasts only three weeks as an average, it probably has been preceded in most cases by several months of tuberculous glandular disease. It has been found that the lower the prevailing temperature the more deaths there are from diseases of the respiratory tract.

As regards the effect of housing conditions in Glasgow, there does not seem to be anything striking. The disease is chiefly in children, and 81.7 per cent of the child population under 5 years live in one or two-room houses. Eighty per cent of the meningitis occurs among these. The remaining 20 per cent of the cases are found among the 18.3 per cent of children living in better houses. The percentage incidence is, therefore, no greater in the overcrowded and poorly housed population.

NEFF, Kansas City, Mo.

ASCITIC TUBERCULOUS PERITONITIS WITH LARGE MASSES. BERNARD MYERS, Proc. Roy. Soc. Med. (Clinical Section) **19**:27 (April) 1926.

The patient, aged 11, on admission into hospital in 1924, was desperately ill with fever and great deal of fluid in the abdomen. Two weeks later the fluid disappeared, leaving enormous nodular masses, which were gradually absorbed. The patient now appears to be in perfect health, and has gained considerably in weight. The treatment was rest in bed, iodine defatted tuberculin and cod liver oil. The author has no reason to believe that the alipoid tubercle bacillary emulsion played any part in the patient's recovery.

WILLIAMSON, New Orleans.

THE DIAGNOSIS OF INCIPIENT TUBERCULOSIS IN CHILDREN. W. H. DONNELLY and H. A. REISMAN, Arch. Pediat. **43**:386, 1926.

The prevalence of a positive tuberculin test is not as universal as was formerly believed. A positive test in a child below 10 years of age is of much significance and should be given due consideration. A persistently negative test indicates the absence of a tuberculous infection. Excluding bone and visceral tuberculosis, tuberculosis in children is essentially a tracheobronchial adenitis, with little or no physical signs except a positive D'Espine's sign. Children with incipient tuberculosis are not necessarily underweight. The incidence of tuberculous infection is not greater among malnourished school children. The rôle that bovine infection plays has been greatly exaggerated with small realization as to the importance of human contagion. From 64 to 81 per cent of school children between the ages of 5 and 14 years, who give a definite history of contact, become infected. A definite history of contact, the presence of enlarged tracheobronchial glands and a positive tuberculin test are sufficient evidence to make a diagnosis of incipient tuberculosis in children.

ULTRAVIOLET RAY THERAPY IN TUBERCULOSIS OF CHILDREN—A RETROSPECT. JOSEPH H. MARCUS, Arch. Pediat. **43**:397, 1926.

The literature on the subject is reviewed. The author's two years experience with the use of the mercury vapor quartz lamp leads him to conclude that the glandular type of tuberculosis is amenable to treatment, the results ranging from fair to excellent. In the latent pulmonary type, the improvement noted was a resultant increase in weight accompanied by a state of euphoria. The existent pathologic change was not influenced. Ultraviolet ray therapy, while not specific, is the most valuable recent contribution to the treatment of certain types of tuberculosis and should not be used as a distinct and separate entity.

VON MEYSENBUG, New Orleans.

THE DEMONSTRATION OF KOCH'S BACILLUS IN THE CEREBROSPINAL FLUID IN TUBERCULOUS MENINGITIS. VITTORE ZAMORANI DOTT, Riv. di clin. pediat. **24**:456, 1926.

The author was successful in culturing tubercle bacilli from the cerebrospinal fluid of twenty-three patients with tuberculous meningitis, whereas in only sixteen (or 70 per cent) were tubercle bacilli found by the usual microscopic methods. A medium of egg was used. It required from eighteen to fifty days for sufficient growth of the bacteria to permit diagnosis.

HIGGINS, Cincinnati.

AMBULATORY TREATMENT OF CONGENITAL LUES. H. OREL and H. WIMBERGER, Wien. klin. Wchnschr. **39**:808 (July 8) 1926; concl., July 15, p. 846.

The authors recognize the disadvantages of antisyphilitic treatment of infants and small children in hospitals, due to unnatural environment and liability to intercurrent disease. Since 1920 they have as far as possible handled such cases in the outpatient department of Pirquet's Univeritäts-Kinderklinik in Vienna. The chief difficulty is lack of cooperation and the abandonment of treatment. This has been combated by conferences with parents, by having the same attendants in charge of the patient throughout, and by use of simple therapeutic agents free from painful or disagreeable after-effects.

Courses of neo-arsphenamine (from 0.1 to 0.3 Gm. intravenously) are given at weekly intervals for twelve weeks. Yellow mercurous iodide (0.01 Gm.,

per os) is administered at home two or three times daily. After two courses, if the Wassermann reaction is still positive, a third is given, and if necessary further treatments after a rest of three months. Patients who have a negative Wassermann reaction after the initial two courses, are allowed a month's rest, then a third course is given, then further blood tests are made after provocative doses of neo-arsphenamine until two years have elapsed. Intramuscular injections are too painful. Stovarsol per os is used in children with nonpatent veins.

Out of 117 patients, 36 per cent were treated thoroughly, 27 per cent incompletely, and 37 per cent were nearly or entirely untreated. Although the total mortality was 24 per cent, excluding deaths from intercurrent disease, it was less than 10 per cent. The outcome of the Wassermann reaction and the development of the children were generally but not always in direct relation to the thoroughness of treatment. A certain individual resistance toward varying degrees of spirochetal virulence plays a rôle. Out of forty-two late syphilitic patients, only one-sixth were clinically entirely normal, and after thorough treatment the Wassermann reaction remained positive in the proportion of 5:3.

Orel and Wimberger, from an economic as well as from a medical standpoint, make a plea for compulsory state control and treatment of all syphilitic subjects.

OLMSTED, Seattle.

DOES MEASLES ACTIVATE TUBERCULOSIS? W. BEISKIN, Ztschr. f. Kinderh. **40**:353, 1925.

In a statistical study of 162 cases, Beiskin confirms the finding of Noeggerath and Eckstein, that there is no direct or frequent activation of tuberculosis by measles. In sixty-four cases of tuberculous meningitis, there was no preceding measles. The curves in the years 1908 to 1924 for the mortality of tuberculosis, of tuberculous meningitis, and of measles did not run at all parallel.

THE QUESTION OF IMMUNITY IN TUBERCULOSIS. N. M. NIKOLAJEW, Ztschr. f. Kinderh. **40**:401, 1925.

Nikolajew, from his clinic in Moscow, analyzes twenty-five cases of infants infected with tuberculosis, judging their resistance to the infection by the amount of cell reaction, that is, the production of epithelioid, connective tissue, and giant cells. Babies with miliary tuberculosis show an outspoken cell reaction, but the mortality is high because the bacilli are encouraged to grow by the slowed lymph stream consequent to the wide lymph sinuses and channels and blood capillaries. No conclusion can be drawn as to the relation of different forms of tuberculosis to immunity. The peculiar course of this disease in infancy can be explained by unspecific conditions relating to their constitution and to their surroundings.

STATISTICS ON CONGENITAL SYPHILIS. HERBERT OREL, Ztschr. f. Kinderh. **40**:414, 1925.

The author shows, in Pirquet's clinic, by a statistical study of 100 pregnancies, in women with untreated syphilis, that sixty living births can be expected, but of these only thirty will live to reach the age of 3 years. When the mother is treated, double that number will reach the third year.

EMERSON, Boston.

THE COURSE OF PULMONARY TUBERCULOSIS AFTER TREATMENT WITH SANOCRYSIN. R. KJER-PETERSEN and V. PETERSEN, Ugesk. f. Læger. **19**:457, 1926.

This paper summarizes the effect of the sanocrysin treatment in seventy-two cases of various degrees of pulmonary tuberculosis. The authors conclude that the treatment is of questionable value. In their opinion, sanocrysin has not helped a greater percentage of patients to reach the arrested stage and become able to work than has the established hospital and sanatorium treatments, nor has it shortened the stay in these institutions. Finally, sanocrysin is disagreeable to many patients, and in occasional instances causes death.

SCHLUTZ, Minneapolis.

Diseases of the Eye

CASE OF DOUBLE OPTIC ATROPHY WITH CEREBRAL AGENESIS. A. FEILING, Proc. Roy. Soc. Med. (Section of Neurology) **19**:8 (April) 1926.

The author reported a case in a boy, aged 26 months, who was well grown and well nourished. He could not sit up, and took no interest in his surroundings. Examination of eyes showed both disks were grayish white. The vessels were of fairly good caliber, with some retinal atrophy. The boy had constant nystagmoid movements of both eyeballs. There was no evidence of paralysis of the limbs.

WILLIAMSON, New Orleans.

Internal Diseases

RECKLINGHAUSEN'S NEUROFIBROMATOSIS AND THE SKELETON. A PLEA FOR COMPLETE STUDY OF THE DISEASE. EDWIN P. LEHMAN, Arch. Dermat. & Syph. **14**:178 (Aug.) 1926.

Attention is called to the fact that bony changes are constantly to be found in cases of Recklinghausen's disease, and that these changes are often of great value in diagnosis. Three types of bone change are to be found (1) scoliosis, (2) abnormalities of growth of individual bones, and (3) irregularity of outline of bones ranging from central and subperiosteal cysts to pedunculated subperiosteal tumors.

JEANS, Iowa City.

NEPHRITIS. N. B. CAPON, Arch. Dis. Child. **1**:141 (June) 1926.

Fifteen cases are reported in detail, together with a brief summary of the usual methods of treatment. A new classification of nephritis is offered.

CHRONIC ULCERATIVE COLITIS. G. BOURNE, Arch. Dis. Child. **1**:175 (June) 1926.

The literature is reviewed and a new case is reported.

PETERMAN, Milwaukee.

INCIPIENT VALVULAR ENDOCARDITIS AS A CLINICAL CONCEPT. NICHOLAS LUKIN, Arch. Pediat. **43**:312, 1926.

A discussion is presented on the pathology and healing process of valvular endocarditis. The possibility of early arrest and limitation of its progress is stated. The formation of a clinical concept of incipent valvular endocarditis with tonsillitis as a primary etiologic factor is urged to that end. The insertion of a special chapter on incipient valvular disease in textbooks is suggested.

VON MEYSENBUG, New Orleans.

THE NECESSITY OF REMOVING BACTERIAL FOCI OF INFECTION IN THE TREATMENT AND PREVENTION OF ENDOCARDITIS IN CHILDREN. MYER SOLIS-COHEN, Arch. Pediat. **43**:423, 1926.

Infecting micro-organisms, rather than diseased tissue, constitute a focus of infection. Removal of diseased tonsils and flushing and aeration of paranasal sinuses do not remove all the infecting micro-organisms from the upper respiratory tract. To eliminate a bacterial focus sufficient antibodies must be developed to destroy the infecting organisms or restrain their growth or neutralize their toxins. Antibody production must be stimulated by the injection of specific antigens. To contain the specific antigens in sufficient quantity, the vaccine must consist chiefly of micro-organisms that are infecting the patient. It is believed that organisms that are pathogenic for a patient and hence infecting him, can be differentiated from organisms that are nonpathogenic for him, to which he stands in the relation of a carrier, by means of the pathogen-selective method of culture. It may be possible in many instances to prevent the occurrence of heart disease by removing bacterial foci of infection in cases of chorea, tonsillitis and other conditions in which endocarditis is recognized as a possible complication.

VON MEYSENBUG, New Oreans.

HABITUAL VOMITING, INANITION AND CONSTIPATION IN A CHILD ASSOCIATED WITH MEGACOLON AND CURED BY BOWEL MANAGEMENT. SARA M. JORDAN, Boston M. & S. J. **194**:1168 (June 24) 1926.

A boy, aged 9 years, had a history of nausea and vomiting, occurring from infancy on, twice a day, usually one hour after meals. There was persistent anorexia, with prolonged eating time up to two hours. Marked constipation was relieved by cathartics and enemas. A roentgen-ray examination revealed megacolon, from cecum to rectum, with direct mechanical pressure on the stomach and upper intestinal tract, and reflex irritation from the extreme dilatation of the colon. "The treatment followed in his case consisted of putting the colon as completely at rest as is possible. The boy remained at rest in bed for three weeks on a diet, low in colonic residue and free of chemical irritants to the colon. Heat and antispasmodic drugs were administered for a time. After four weeks no further medication was given, and cathartics or enemas have never been given since the beginning of treatment." The child gained at the rate of 2 pounds weekly at first, and after thirteen months has gained 32 pounds (14.5 Kg.), the bowel function is normal, and the general condition is that of a well nourished, healthy, fun-loving boy.

GENGENBACH, Denver.

PITUITARY DISEASE IN A BOY AGED FOUR. SIGURD W. JOHNSEN, J. A. M. A. **86**:3 (Jan. 2) 1926.

At the age of 4 the boy weighed 65 pounds (29.5 Kg.) and measured 125 cm. long. The penis was unusually well developed. Roentgen-ray finding showed an enlarged sella turcica with the posterior clinoid process almost vertical. The skeletal development corresponded to that of a 14 year old person.

THE SIGNIFICANCE OF CARDIAC EXTRASYSTOLES IN CHILDHOOD. MURRAY H. BASS, J. A. M. A. **86**:387 (Jan. 6) 1926.

Three varieties of extrasystoles occur in childhood, namely, emotional, toxic and idiopathic, the last two being the most important. The toxic variety, which occurs during or as the result of acute infections, is usually transitory, and,

coincident with acute cardiac inflammation, is not necessarily indicative of severe permanent cardiac injury. As a rule, irregularity completely disappears and does not tend to recur. The idiopathic form is seen in the healthy child, appearing without cause. This condition is more or less permanent and does not seem to predispose to, or result in, cardiac weakness. Children who show extrasystolic contractions every few beats over a period of years need not present any other subjective or objective symptoms of cardiac disease. The five cases reported lead the author to conclude that extrasystoles are not of particularly serious import when they occur in childhood.

SINUS RESPIRATORY ARRHYTHMIA IN CHILDREN WITH RHEUMATIC HEART DISEASE. FRANCIS H. MCCRUDDEN, J. A. M. A. **86**:535 (Feb. 20) 1926.

The author's observations on children with rheumatic heart disease do not confirm the opinion that sinus respiratory arrhythmia is absent only in the most serious cases of heart disease. Analysis of 100 cases was made. Twenty were without arrhythmia, and of these seven died within a year; seven more developed complications or further heart injury or became worse in some way after discharge, and had to be readmitted to the hospital; two more were getting worse; only one was doing well, and the other three did not return. Of the eighty children with arrhythmia, none were known to have died, and none returned to the hospital or became worse. No special follow-up efforts were made.

BONAR, Salt Lake City.

TREATMENT OF IDIOPATHIC PURPURA HEMORRHAGICA BY EXPOSURE TO MERCURY VAPOR QUARTZ LAMP: PRELIMINARY REPORT. J. W. SOOY and T. S. MOISE, J. A. M. A. **87**:94 (July 10) 1926.

The experimental work of Laurens and Sooy has demonstrated a definite increase of the blood platelets after irradiation of white rats with the mercury vapor quartz lamp. A rational basis for the application of this method is offered therefore in diseases associated with platelet deficiency.

The authors report a case of purpura hemorrhagica in which quartz light therapy was beneficial. The treatment was given to the whole body beginning with six minutes' exposure at a distance of 13 inches and increasing the exposure time gradually to twenty-eight minutes. Daily treatments were given for a period of three weeks. Besides purpura hemorrhagica, the authors have had satisfactory results from this treatment in hemorrhagic disease of the new-born and in the preoperative preparation of jaundiced patients.

[ARCH. DERMAT. & SYPH.]

CELIAC DISEASE. F. MOORE, J. Iowa M. Soc. **16**:273 (June) 1926.

This disease is a form of chronic intestinal indigestion, characterized by extreme intolerance for carbohydrates and fats. Moore believes its importance is out of proportion to its frequency. He gives a good review of literature and cites Gee's description as part of intestinal symptoms: "There is a kind of chronic indigestion which is met in persons of all ages, yet is especially apt to affect children between 1 and 5 years of age. The signs of the disease are loose, unformed but not watery feces, more bulky than the food taken would seem to account for: pale as if devoid of bile; yeastly, frothy, and the stench often very great. The pale, loose stool looks very much like oatmeal porridge or gruel."

Mention is made of similarity of sprue of adults and celiac disease of childhood. The onset is often insidious. Failure to grow and fluctuation in weight are striking characteristics; loss of weight is often quite marked. Relapses show marked loss of weight; the body and extremities are thin but the face may seem plump. Pallor and irritability are usually present. The characteristic pathologic changes are unknown. Necropsy changes are reported differently by different observers.

Medical treatment is secondary to diet and is wholly symptomatic. The diet is the most important consideration in treatment. The basic food requirements for maintenance, for growth and energy should be considered; give largely of proteins with the minimum amount of carbohydrates and fats; fresh milk is contraindicated; dried milk or fat free lactic acid milk may be given, also Jello, banana, egg, concentrated beef juice, orange juice, arrowroot crackers, dextrinized starches, etc. Early appropriate treatment is essential.

TURNER, Des Moines, Ia.

THE PREVENTION OF HEART DISEASE IN CHILDHOOD. HARROLD A. BACHMANN, Med. Clin. N. Am. **9**:1531 (May) 1926.

About one third of the author's cases of chorea eventually developed heart lesions. The likelihood of cardiac involvement is directly proportionate to the frequency of the attacks, and the presence of complicating rheumatism and tonsillitis. The most essential features of treatment are rest and avoidance of excitement. Large doses of salicylates, of which the author's choice is acetyl salicylic acid, seem to benefit some cases. He is skeptical about the value of arsenic.

Rheumatism appears to be less frequent among children of the more intelligent classes. This observation emphasizes the value of preventive measures, and suggests that the ultimate solution of the problem of the child with cardiac trouble lies in this direction. Typical acute rheumatism is rather rare in childhood. Muscular pain and soreness, and "growing pains" are more common. The degree of cardiac involvement, however, is in direct relation to the severity of the rheumatic symptoms. Extensive heart damage is always to be expected when subcutaneous nodes develop. A common cold almost invariably precedes a rheumatic attack. Absolute rest after all acute symptoms have subsided is imperative. Tonsillectomy is not a panacea, but is often useful.

Once heart disease is established, information as to what the heart can do is of more value as a guide to treatment than the sounds it produces. During the acute phase, rest must be enforced until all active symptoms have disappeared. The original sources of infection should be eliminated if possible. Later, the health and tonicity of the heart muscle should be developed by carefully supervised exercise and play. The patient must be strictly guarded against new infections, since these are more often the cause of decompensation than exercise. Nose and throat colds are the greatest menace of the child with cardiac disease. In the common cold "lie all the potentialities of most of the serious medical conditions observed in childhood."

GASTRO-INTESTINAL HEMORRHAGE IN CHILDREN. JULIUS H. HESS and PHILIP ROSENBLUM, M. Clin. N. Am. **9**:1577 (May) 1926.

Case 1.—A duodenal ulcer in a girl, aged 8½ years, was reported. After forty-eight hours of cramp-like pains without vomiting or gastric symptoms, the first indications of blood in the stools were noticed. A few days later the patient suddenly fainted, became pale and passed a black stool. There was

no history of any preceding gastro-intestinal disturbance. Roentgen-ray examination showed a typical ulcer defect in the bulbus duodeni. Since the institution of strict ulcer treatment, there has been no recurrence of the pain or hemorrhage, and during the year that has elapsed, the child's general condition has improved markedly.

Case 2.—Congenital atresia of the duodenum by adhesions was reported in an infant, aged 10½ months, who had vomited more or less since birth. This symptom was relieved somewhat by atropine, but was never absent for longer than a few days at a time. A short time before admission, vomiting became more frequent, and a cough and slight fever developed. There was visible peristalsis of the stomach, dehydration and blood in the stool. A roentgenogram showed 95 per cent retention after four hours. Later, the vomitus contained large quantities of brownish material, and brown, watery, frequent stools were passed. At operation the pylorus was found to be patent, and was 12 cm. in diameter. The duodenum was about 8 cm. in diameter, and was constricted about 3 inches below the pyloric ring by adhesions to the posterior abdominal wall and liver. Gastro-enterostomy and pyloric closure were performed. Hemorrhage was probably due to venous congestion. The child recovered.

Case 3.—Bleeding Meckel's diverticulum was reported in a well nourished boy, aged 11 months, who had hemorrhage from the rectum, at first without other symptoms. About two months later there was recurrence of the hemorrhage, with straining, fainting, fever, vomiting, loss of weight and anemia. Operation disclosed a Meckel's diverticulum with ulceration. Recovery was prompt after its removal.

Case 4.—Splenic anemia was reported in a previously healthy girl, aged 14 months, who became less playful, drowsy and pale, and two or three days later vomited about 6 ounces of blood and passed tarry stools. The liver was not palpable and the spleen only slightly so. Blood examination showed, hemoglobin, 30 per cent; red blood cells, 2,600,000; white blood cells, 13,000; neutrophils, 60; small mononuclears, 34; large mononuclears, 4; unclassified, 2. Improvement followed transfusion. Several months later the patient was again passing blood and had fever with increasing anemia. The spleen was large and firm. Splenectomy was performed, and a diagnosis of fibrotic splenomegaly (Banti's disease) was confirmed. Steady improvement followed the operation. About two months later, blood examination showed, hemoglobin, 60 per cent; red blood cells, 4,900,000; white blood cells, 14,000; polymorphonuclears, 20; small mononuclears, 70; large mononuclears, 6; transitionals, 4.

SMITH, Ogden, Utah.

ETIOLOGY AND TREATMENT OF ENURESIS. M. K. WYLDER, M. J. & Rec. **123**:358 (March 17) 1926.

Enuresis is not a disease, but only a symptom caused by many conditions of widely divergent pathology. The causes given by various authors are discussed. The many and varied causes given emphasizes the fact that there is not at the present any very definite idea as to the cause. The general idea prevailing is that anything that disturbs the mental or physical condition of the patient may give rise to this symptom. Many methods of treatment are given. The points on which all recent authors agree are these; no fluids after

4 p. m., a rest at midday; empty the bladder before retiring and again before midnight; encourage the child and impress on him that he can get well. Use a chart with gold stars for dry nights and red ones for wet nights, this stimulates the subconscious control over the higher centers to greater activity and strengthens the will. Drugs are more psychologic than medicinal.

WILLIAMSON, New Orleans.

CHRONIC ABDOMINAL PAIN IN CHILDREN. KIRSTEN UTHEIM TOVERUD, Brit. J. Child. Dis. **23**:22 (Jan.-March) 1926.

The author summarizes his study as follows:

Chronic constipation in some children will sooner or later give rise to a picture which may suggest a severe disease. Recurrent abdominal pain is the most predominant feature of this condition, besides general malaise. The condition covers mostly what in different paediatric textbooks has been described under the name of "colitis," "chronic gastric catarrh," or "intestinal colic." From the material at the Pediatric Clinic of the State University of Norway, the cause seems to be mostly poor food and the child's bad habits. A change in the food to the so-called constipation diet, and a change in the child's habits have been able to cure these children completely.

OBSERVATIONS ON GOITER IN CHILDREN. H. LESLIE CRONK, Brit. J. Child. Dis. **23**:32 (Jan.-March) 1926.

According to Cronk, the cause of goiter in children may be:

1. An hereditary or early acquired diminution in secretory activity of the cells of the thyroid.

2. The relative increase of weight of the body, causing increased need of thyroxin for metabolism.

3. Some factor which affects the tonsils in such a manner that the rise in prevalence of enlarged tonsils coincides with a diminution of goiter prevalence, and exerts such an effect on the latter that the enlarged tonsil rate at times overrides the effect of increased metabolism needed for increased body weight.

There is at present no clue as to the nature of the last factor.

PNEUMOTHORAX IN YOUNG CHILDREN, WITH AN ACCOUNT OF A CASE. ALAN MONCRIEFF, Brit. J. Child. Dis. **23**:37 (Jan.-March) 1926.

The author cites a case of right-sided pneumothorax in a boy, aged 1 year and 10 months. The favorable outcome and the negative Pirquet test both suggested that the case was not tuberculous in origin, and was probably caused by the rupture of a subpleural emphysematous vesicle in the course of a bronchopneumonic process in the right lung.

The author mentions the findings of various others in regard to pneumothorax. Cruchet estimated only 40 per cent of all cases of pneumothorax in children as due to tuberculosis. Moncrieff calls attention to the fact that, except for Sutherland's article in Garrod, Batten and Thursfield's textbook, most textbooks on diseases of children make only very brief mention of pneumothorax.

LANGMANN, New York.

FREQUENCY OF MICTURITION IN INFANCY AND CHILDHOOD. DONALD PATERSON, Practitioner **116**:696, 1926.

Due to liquid diet under 2 years, frequency is not pathologic. Attempts should be made to teach the infant control, however, from the age of 1 month. Success depends largely on the fidelity of the nurse in charge.

The causes of frequency are divided into those in which the bladder or urine are at fault, and those in which training or nervous control of the bladder are at fault.

Cystitis and stone are very rare, although pus and organisms are frequently encountered. Occasionally, very acid urine is at fault, and citrate of potash or bicarbonate of soda are indicated. If the amount of urine passed is very great diseases such as interstitial nephritis and diabetes of one or the other type must be excluded.

In cold weather there is always a great diuresis. During the teething period, frequency occurs, but this is without adequate explanation, although it may be due to anorexia, more fluid food, greater acidity of the urine. If these points have been ruled out, it should be assumed that the fault lies in the nervous control of the bladder.

Attempts should be made to teach the child to hold a larger amount of urine in the bladder before evacuation. This may be accomplished by distracting his attention over the period when he would urinate. Discussion before the child should be carefully avoided. Any mention to the child should be one of encouragement, not reproof. Frequency may develop following illness due to a debilitated condition, which is cured as the child's nutrition is improved.

Nervous children are very subject to frequency, particularly during periods of excitement, such as holidays, change of nurses, etc.

The only drug of value is belladonna or atropine. This is given as the tincture of belladonna, well disguised in syrup of orange, three times a day before meals.

It is of interest that in cases of enuresis, it was noted that frequency occurred in the day in three out of every four cases.

Mentally defective children and those with organic nervous disease are not considered.

TREATMENT OF CHILDREN RECOVERING FROM ACUTE CARDIAC AFFECTIONS. LESLIE THORNE THORNE, Practitioner **117**:29, 1926.

The data on which this article is secured are from two convalescent homes, one for boys and one for girls. It is to demonstrate the value of the Nauheim treatment for children suffering from either acute or subacute cardiac disease, rheumatic in origin or otherwise. For instance, whooping cough, measles, scarlet fever, etc.

The cardiac defect resulting from whooping cough is apparently a myocardial damage based on a previous rheumatic infection. It often happens that myocarditis is diagnosed with great difficulty, and its symptoms are confused with those of anemia. For this reason children are given tonics and are recommended to take exercise which, of course, tends to aggravate the condition.

For a diagnosis of cardiac dilatation in its early stages, Poynton relies almost exclusively on percussion of the deep cardiac dulness. The author agrees with this point of view, and feels that the electrocardiograph is of very little value in the early stages of myocardial damage.

Absolute rest in bed is essential in the early stages of the treatment, combined with the Nauheim baths. A course of from 18 to 25 baths are given, extending from four to five weeks which, of course, is a much shorter period than is usually necessary. Two case histories are given as illustrations.

GELSTON, San Francisco.

DISCUSSION ON THE DIAGNOSIS AND TREATMENT OF SPLENIC ENLARGEMENT IN CHILDREN. SIR HUMPHRY ROLLESTON, Proc. Roy. Soc. Med. (Section for the Study of Disease in Children) **19**:19 (Feb.) 1926.

Enlargement of the spleen is a physical sign due to many causes which in the vast majority of cases are infective. The spleen and lymphoid tissues generally are more prone to active response and enlargement in childhood than in later life.

It is of interest to note that in chronic diseases (in which it is usual for the spleen to enlarge), splenic enlargement is elicited in any early stage of the disease when no other symptom is showing. Splenomegaly may constitute the only clinical sign of an incomplete form of a disease, such as Hanot's cirrhosis or the only physical sign in a member of a family in which other members had Hanot's hypertrophic biliary cirrhosis (Boinet). Again, splenic enlargement may be due to more than one factor.

The author groups the forms of splenomegaly under a number of inclusive headings:

1. Familial, that is, Gaucher's disease, congenital syphilis, and chronic splenomegalic hemolytic jaundice.
2. Those associated with changes in the blood, that is, anemia, leukemia.
3. Infective.
4. Those of tropical origin.
5. Mechanical.
6. Those associated with hepatic enlargement.

He then discusses the various diseases under these headings.

SPLENECTOMY FOR ESSENTIAL THROMBOCYTOPENIC PURPURA HEMORRHAGICA IN A GIRL AGED TEN YEARS. BERNARD MYERS, Proc. Roy. Soc. Med. (Clinical Section) **19**:31 (May) 1926.

The patient was admitted to hospital on account of bleeding from the mouth and nose. There were numerous small bruises and many petechiae on the body. A small amount of blood was present in the urine on one occasion. The spleen was not palpable on admission, but was distinctly felt four months later.

The blood report was as follows: Red blood cells, 4,900,000; hemoglobin, 50 per cent; color index, 0.5; leukocytes, 16,250; polymorphonuclears, 59 per cent; lymphocytes, 38 per cent; the platelets were fairly numerous; anisocytosis was present. As the usual treatment was quite unavailing, splenectomy was performed with excellent results. It is first necessary, however, to make sure that the case comes within the category of those suitable for operation. The preliminary tests consist of: (1) The presence of thrombocytopenia; (2) prolonged bleeding time; (3) normal clotting time; (4) a positive capillary pressure test. The operation was carried out by Rodney Maingot, F.R.C.S., and the details are given in full.

WILLIAMSON, New Orleans.

DIABETES INSIPIDUS OF SYPHILITIC ORIGIN. J. BABONNEIX and J. LHERMITTE, Ann. de méd. **18**:471, 1925.

This is a report of a case of polydypsia and polyuria. The necropsy revealed a localized leptomeningitis in the region of the optic chiasm and the tuber cinereum. There were perivascular infiltrations and acute hemorrhages in the infundibulum and the tuber region. The paraventricular and supra-optic nuclei were normal, but the cells of the tuber nuclei were reduced in number and showed degenerative signs when stained with Nissl's method.

WEIL, New York.

CLINICAL AND BLOOD CHEMISTRY STUDIES ON GENUINE INFANTILE LIPOIDAL NEPHROSIS. G. DETONI, Clin. pediat. **8**:212-226, 1926.

Lipoidal nephrosis belongs to the degenerative types of diseases attacking the parenchymatous structure of the kidney. It is not caused by chemical poisons or bacterial toxins. It is commonly associated with chronic constitutional diseases. Syphilis gives rise to the purest form of this condition. In tuberculosis it is frequently mixed with an amyloidosis. There is also an essential or genuine lipoidal nephrosis the etiology of which is unknown. It is due to a general disturbance of lipoid metabolism as indicated by the marked hypercholesterinemia and lipoiduria.

It has a rather distinctive clinical picture. Edema and more frequently anasarca is a very prominent feature. It resists all therapy and is even aggravated by the strictly milk diet. This is due to the fact that the fluid remains in the tissues because of a physiochemical change in all the body colloids. The elimination of nitrogenous substances in the urine is normal, and there is therefore no danger of uremia. There is no hypertension or cardiac hypertrophy as seen in the sclerotic lesions of the kidney. Albuminuria is very pronounced; it ranges from 4 to 50 per cent. The urinary output for twenty-four hours is strikingly diminished (250 to 400 cc.). The urine is usually turbid and the specific gravity is from 1,030 to 1,050. Microscopic examination of the sediment shows it to be large in amount and contains no gross blood. Hematuria is not seen as a rule. Microscopically, there may be a few scattered red blood cells and casts. Lipoid granules, bi-refracting on polariscopic examination, are always present in large quantities. They may be intracellular or extracellular. The disease pursues a very chronic but benign course with numerous remissions. The patients often dies of some intercurrent infection.

A case is reported in a child, aged 3½ years. The patient had an intense anasarca, which resisted all dietetic, physical and pharmacologic treatment. He died of bronchopneumonia two months after the appearance of renal disease. The urinary output for twenty-four hours ranged from 200 to 350 cc. The specific gravity was from 1,035 to 1,044. The very abundant sediment contained a very large amount of albumin. There were many hyaline and granular casts, and a very occasional red blood cell. Bi-refracting lipoid masses were seen on polariscopic examination. The blood pressure varied from 110 to 70. It was not influenced by the administration of urea. Blood chemistry estimations revealed normal values for urea, nonprotein nitrogen, creatinine, calcium and chlorides. There was a moderate hyperglycemia and a very pronounced hypercholesterinemia.

Therapy is futile and the disease itself is benign, but the fatal intercurrent infections find a suitable soil in an organism whose metabolism is so profoundly altered and whose resistance is lowered.

ZAMBARANO, Northville, Mich.

ARSENICAL POLYNEURITIS IN THE CHILD. LUIS MORQUIO, Arch. españ. de pediat. **10**:257, 1926.

This report concerns nine children, five of one family and four of another, who were suffering from arsenic poisoning due to ingestion of food containing it. They developed all the acute gastro-intestinal symptoms during the acute period and, in addition, sensory and motor phenomena characteristic of this poisoning, leading finally to paralysis of the extremities.

In the first group of cases the symptoms of poisoning set in immediately after a meal and developed in two stages. The primary immediate symptoms were gastro-intestinal and lasted for several days. These were followed two weeks later by motor symptoms. In the second group of cases the poisoning occurred more slowly. Motor symptoms set in within about fifteen days and were accompanied by slight gastro-intestinal symptoms. All motor phenomena were progressive, and rapidly resulted in complete paralysis.

Paralysis due to arsenic poisoning has several striking characteristics. It affects the extremities exclusively, particularly the legs, and favors the extensor groups. There is pain and loss of tactile sensation in the affected parts. The lesions are always symmetrical. There is discrete and irregular atrophy. The reaction of degeneration is quite incomplete. The patellar reflexes are lost. There is trophic disturbance of the skin and nails, with presence of melanodermia. All sphincter control is maintained and there is complete absence of any meningeal manifestation.

The tendency is toward recovery, particularly if properly treated. The period of recovery generally extends over several months. The treatment consisted in the use of massage, warm baths and the use of faradic current. Iodides were given internally.

REPORT OF A CASE OF DEXTROCARDIA. SORIA JOAQUIN, Arch. españ. de pediat. **10**:347, 1926.

The author reports the case of a boy, aged 12, with dextrocardia without transposition of any of the other viscera. The author raises the question whether the transposition was congenital or possibly acquired. Congenital dextrocardias are practically always accompanied by transposition of other abdominal viscera. On account of the age of the child, and the absence of any known chest pathologic changes except a moderately severe attack of pneumonia, the author concludes that the dextrocardia is congenital in origin.

COMPENSATORY COLLATERAL CIRCULATION IN A CASE OF CONGENITAL PULMONARY STENOSIS. W. ARNOLD, Centralbl. f. allg. Pathol. u. path. Anat. **37**:193, 1926.

The collateral circulation consisted of a superior bronchial artery arising from the concave part of the aortic arch, a branch of the left subclavian artery, a posterior bronchial artery springing from the descending part of the aort and two arteries probably coming from the inferior thyroid artery. Between these vessels there were numerous anastomoses. The right lung was ultimately supplied by four arteries and the left by three.

GEORGE RUKSTINAT.

ENDOCARDITIS IN EARLY CHILDHOOD. R. GEIGER, Ztschr. f. Kinderh. **40**:370, 1925.

Geiger presents a table of ninety cases of acquired endocarditis in children up to 4 years of age collected from the literature since 1844, and adds to this a series of eighteen collected in Munich in the last twenty-five years. There are several points of interest: 1. In the institutional cases years ago, there were few cases of endocarditis reported, and considering the number of cases of rheumatic fever and of chorea, many must have been overlooked. 2. The incidence of endocarditis is remarkably low in institutions in Europe, quite as surprising to us as the large number of cases reported in the United States is to Geiger. 3. There are two grades of morbidity, one for 6 to 18 months, in whom the cause seems to be grip and sepsis, and another at 4 years, associated with rheumatic fever. 5. The outcome of children up to 4 years is almost always, for babies always, fatal. The duration of illness for children up to 4 years is six months at the outside; for babies, a few weeks. One cannot agree with the author that the symptoms of rheumatic fever in the first four years are so pronounced that they cannot be overlooked.

EMERSON, Boston.

Nervous Diseases

THE NATURE OF THE FUNDAMENTAL DRIVES. EDWARD CHASE TOLMAN, J. Abnorm. Psychol. **20**:349 (Jan.) 1926.

This is an interesting article giving first the criteria for determining the characteristics of a fundamental drive: (1) individuals of a species must all exhibit a recurring tendency toward certain ends; (2) the tendency toward the need must appear in behavior otherwise different; (3) the difference of strength (of drive) explains difference of behavior at different times in different persons. What seems a departure from the title is dividing the drives into those of primary and secondary orders. The primary drives are those correlated to a physiologic need — hunger, sex, fatigue, excretion and sensitive zones. These are spoken of as appetites. The aversions, also of the primary order, are fear and pugnacity. Drives of secondary order, gross behavior characters, are: curiosity, gregariousness, demand for dominance and demands of submission and imitativeness. The drives of the secondary order are measurable and represent responsiveness to early environment and training. Their dependence on the primary truths must also be considered.

CHARACTERISTICS OF THE NEUROTICALLY PREDISPOSED. W. S. TAYLOR, J. Abnorm. Psychol. **20**:377 (Jan.) 1926.

This is a synopsis of an article by Dr. A. F. Riggs on the psychoneurotic patient: who is physically well above the average; whose environment contains no typical difficulty, and yet who is inefficient. The psychoneurotic is oversensitive to emotion and sensation. His instinct of self preservation is overactive with consequent prominence of fear and anger; he is overly suggestible and has character faults usually from a lack of training and discipline. He suffers from a "relative unbalance" of instinct. This leads to a dissociation and inefficiency. Being in the nature of an abstract of another article, "The Psychoneuroses: Their Nature and Treatment" (*Am. Jour. Psychiat.* **80**:94-109, 1923) further abstraction is impossible. The original article must be read by any one wishing the real substance.

HAMILL, Chicago.

THE DEVELOPMENT OF SPEECH IN THE NORMAL CHILD. MACLEOD YEARSLEY, Brit. J. Child. Dis. **23**:1 (Jan.-March) 1926.

The author traces carefully the development of speech in his own child, and frequently alludes to a similar study made by Preyer. He finds that the literature on the subject is ridiculously meager.

In summing up, he states that we may fairly consider that the first three years of the normal child's life may, in regard to the development of speech, be apportioned as follows, always remembering that there is of necessity a certain amount of overlapping.

The first year is spent in methods of communication which are part animal, part primitive human, these methods serving as a kind of scaffolding beneath which the main building of articulate speech is being built up. The second year is devoted to the compilation of a vocabulary and experiments therewith, the third year being occupied in perfecting and correcting. These years of speech development are practically an epitome of the evolution of language.

LANGMANN, New York.

CASE OF DISORDERED RESPIRATORY RATE AND INVOLUNTARY CRYING; AFTER-EFFECTS OF EPIDEMIC ENCEPHALITIS. W. G. WYLLIE, Proc. Roy. Soc. Med. (Section of Neurology) **19**:8 (April) 1926.

A girl, aged 13 years, eighteen months prior to examination suffered from insomnia and headaches, and appeared to make a good recovery.

For the last six months, the patient had suffered from attacks of rapid and shallow breathing, often losing consciousness, accompanied by tonic contraction of the limbs ending with a brief spell of crying. There was considerable emaciation.

CASE OF WRIST-DROP IN A CHILD. G. A. SUTHERLAND, Proc. Roy. Soc. Med. (Section for the Study of Disease in Children) **19**:48 (May) 1926.

The patient, a boy, aged 8 years, was admitted to hospital suffering from acute nephritis, right epididymitis, and a purpuric rash on the feet. Later, a right lobar pneumonia developed, then a left. The child made a good recovery. A month after, he was found to have a left wrist-drop. There was some weakness of the right hand. The temperament of the boy was highly neurotic. The question is, what is the cause of the wrist-drop? For the purpose of discussion, the author regarded it as functional paralysis.

WILLIAMSON, New Orleans.

THE RÔLE OF THE HYPOPHYSIS IN INFANTILE DYSTROPHIES. P. LEREBOULLET, Ann. de méd. **18**:452, 1925.

This is a critical review of recent literature. The so-called hypophysis syndromes in children are often produced by lesions of hypothalamic nuclei. Only gigantism and acromegaly may now be considered as pituitary diseases.

WEIL, New York.

HEMORRHAGIC PACHYMENINGITIS IN INFANTS. R. DEBRÉ and G. SEMELAIGNE, Presse méd. **34**:337 (March) 1926.

The authors report five certain and two probable cases of hemorrhagic pachymeningitis in infants, seen in one year. The usual age is 2 to 6 months. The onset is insidious, with weakness, insomnia, anorexia, and indefinite

nervous symptoms, but occasionally is sudden with high fever and convulsions. Hydrocephalus is one of the earliest signs. The diagnosis is made by puncture of the ventricle and ophthalmoscopic examination. The fluid is under increased pressure, red or yellow, and after it has stood, red blood cells separate out. Cultures are sterile. The fluid from the spinal canal is normal or merely yellowish. In about half of the cases there are retinal hemorrhages. The course is usually subacute, and the patients that do not die of intercurrent events make a gradual recovery. A few die of the meningitis itself, usually the acute form. Sequelae sometimes remain. The essential lesion seems to be the formation of a false membrane, with secondary hemorrhage from the altered vessels. Signs of congenital syphilis are present in many cases. Antisyphilitic treatment is advised, with ventricular puncture, injection of coagulants, and occasionally cerebral decompression.

B. R. Lovett.

Bradycardia in Children. Case of Congenital Heart Block Associated with the Morgagni-Adams-Stokes Syndrome. G. DiToni, Clin. pediat. **8**:65-98, 1926.

The author reports the case of a boy, aged 8 years, with Adams-Stokes disease. In the present state of our knowledge, infantile bradycardias cannot be subdivided etiologically, but can be classified clinically. Permanent bradycardia may be complete or incomplete. Acquired total bradycardia has been observed in children suffering from appendicitis, chronic spastic colitis, icterus, tuberculous meningitis and in all morbid processes exerting toxic influences on the vagus and its branches. A differential diagnosis between the total bradycardias and other forms of dissociation is possible only by resorting to graphic methods. The Morgagni-Adams-Stokes syndrome may coexist indifferently with a disturbed conduction (complete or incomplete). If complete dissociation is present, the influence of the extracardiac nervous agencies on the ventricular automatic rhythmicity, cannot be lost sight of. This was shown in the case reported. Stimulation of the vagus caused syncope. Interpretation of this fact is yet unsatisfactory.

Functional examination of the extra cardiac nervous influences, particularly the autonomic, is always necessary even in cases of permanent total block.

The Reducing Power and the Pressure of the Cerebrospinal Fluid in Pediatrics. A. Baccichetti, Clin. pediat. **8**:129-150, 1926.

The author made a careful and exhaustive study of the reducing power and pressure of the spinal fluid in 168 patients, the majority of whom were suffering from diseases of the nervous system. He found that the reducing substances in the spinal fluid are not all glucose or its derivatives, but other substances probably arising from nerve cell metabolism and cellular exchange. In normal infants the reducing power, determined by the Pavy-Sahli method, lies between 0.48 and 0.60 per thousand; 0.56 under 1 year of age and 0.51 over 1 year of age. The normal pressure determined with the Claude manometer rests between 25 and 30 mm.

In tuberculous meningitis, the reducing power is always diminished even when the clinical symptoms are poorly defined. In 50 per cent of the cases it disappears entirely. This absence of the reducing power is observed in 69 per cent of the cases occurring in infants under 1 year of age, and in 33 per cent of those past the first year of life. The pressure of the fluid in this condition is always increased.

In purulent meningitis, the power of reduction is diminished and is absent in 58 per cent of the cases.

The power of reduction in serous meningitis is always retained and at times is increased. In hydrocephalus and Little's disease, it is increased if they are of syphilitic origin; in the cases that are sequelae of meningitis it is normal or slightly diminished.

Encephalitis lethargica retains the reducing power of the spinal fluid; in 80 per cent of the cases there is a slight increase. The acute stage of Heine-Medin disease is accompanied by an increased reducing power; with the onset of paralysis it returns to normal. The reducing power is normal in rheumatic chorea, pertussis, postdiphtheritic paralysis, pulmonary tuberculosis, congenital syphilis without hydrocephalus, gastro-enteritis and rickets. In cases of miliary tuberculosis with no symptoms referable to the meninges, the reducing power is diminished. The pressure of the spinal fluid showed so much variation in each case that it is impossible to derive any conclusions. The pressure in cases of brain tumor in infants is not relatively as high as it is in adults.

PATHOLOGIC ANATOMY OF ENCEPHALITIS EPIDEMICA IN CHILDREN. F. ZIBORDI, Clin. pediat. **8**:151-160, 1926.

Suspecting his three cases to be of influenzal origin, the author gave his patients an intradermal injection of a vaccine containing about ten million Pfeiffer bacilli as a diagnostic test. For control, he injected also vaccines containing staphylococci, streptococci and pneumococci in the same amount. After twenty-four hours, the characteristic red zone of a positive reaction appeared around the area at which the influenza vaccine was introduced; the controls provoked no response. This phenomenon favors the view that there is some etiologic identity between influenza and epidemic encephalitis. He gives a very lengthy and detailed necropsy report of one of his cases. The microscopic anatomy was studied very carefully. In brief, he found the brain congested and edematous. There were numerous small hemorrhages scattered throughout the vault. There was marked vascular dilatation and perivascular lymphocytic infiltration throughout the entire cerebrum but chiefly in the cortex and optic thalamus.

DIAGNOSIS AND TREATMENT OF POSTDIPHTHERITIC PARALYSIS. P. FORNARA, Clin. pediat. **8**:228-240, 1926.

In one of the cases of postdiphtheritic paralysis reported, the symptomatology simulated epidemic encephalitis, especially the somnolence, intellectual torpor, nystagmiform movements, tremors and myoclonus. These lead the author to believe (as do other observers) that there may be some central lesion in the postdiphtheritic paralyses. It may be primarily a nuclear involvement or a secondary ascending intoxication. In discussing the prophylactic and curative value of antidiphtheria serum in postdiphtheritic paralysis, eight illustrative cases are presented. In three, during the stage of angina, no serum therapy was used. In two the amount used was insufficient, and in three other cases intensive treatment was given but too late. In none of the cases, therefore, was ample serum given to prevent the onset of paralysis.

Massive doses of serum were given (30,000 units) to four cases as curative treatment for the paralysis. Good results were obtained in all cases. None of the patients had very severe paralysis however. Four other cases were treated with ordinary measures, strychnine and electrotherapy. Progress was much slower but a cure was finally attained.

Another case is reported, in which the patient received 45,000 units subcutaneously and intramuscularly and 20,000 units intraspinally. The immediate result was good and the patient appeared much improved. On the sixth day following the intraspinal injection, signs of meningitis appeared and the child died. The spinal fluid twenty-four hours after the injection was purulent. The meningitis was probably caused by irritation of the injected serum or it was the expression of an anaphylaxis. The author urges that all serum therapy be administered intramuscularly, subcutaneously or intravenously.

ZAMBARANO, Northville, Mich.

ACANTHOSIS NIGRICANS IN IDIOCY. LADISLAUS BENEDEK and KARL CSÖRSZ, Jahrb. f. Psychiat. u. Neurol. **44**:291, 1925.

Acanthosis nigricans is characterized by an ichthyosis and condylomatous hypertrophy of the corneal and papillary layers of the epidermis, associated with an abnormal deposit of pigment (dystrophie papillaire et pigmentaire), and occurs most commonly in cases of malignant tumor of the internal organs. This unusually rare skin disease is usually found on the neck, face, hands, axillae and genitofemoral region; it is also commonly associated with other trophic disturbances. The affected skin presents small, club-shaped, dark brown, confluent nodules with pale rose colored streaks of healthy skin between them. Bogrov could find only fifty-one cases reported in the literature up to 1908. Darier's juvenile type may, according to Jessner, also occur without tumors of the internal organs.

Parhon and Dan described ichthyosis of the skin in congenital idiocy associated with bradycardia and mononucleosis. Recently, Goldblatt observed acanthosis nigricans in a case of mental debility, and Filsen (1908) found it in a patient with epilepsy and stuttering. In Goldblatt's case the idiocy was not of high degree; his patient was an Ukrainian peasant, who was able to take an active part in the World War, and who, owing to periodic episodes of excitement brought on by his war experiences, came under medical observation for his mental condition.

The case reported by the present authors occurred in a man, aged 20, born in Debrecen. There was no history of stuttering, bed-wetting, migraine or a similar disease in the family. A maternal cousin (female) is left-handed; his parents are healthy; alcoholism and syphilis are denied. The patient's mother had given birth to nine other children; one child was stillborn; five died of convulsions between the ages of 6 and 12 months; the remaining three children are in good health.

The patient's birth was uneventful; teething began at 3, walking at 5 and talking at 7 years of age. He attended school but could not learn to read or write a single letter. According to the mother his interest in his surroundings and his fund of general information was greater when he was going to school than now. There are no reliable data as to what children's diseases he had. In his third year he began to have convulsions; these were always preceded by greater pallor and were bilateral; during them he would scream and foam from the mouth, but he never lost sphincteric control. The attacks would usually come on during the night and last several minutes. Between the ages of 3 and 7 he would get an attack every other night. The attacks became less frequent after the age of 7; he would sometimes be free from attacks for two or three years. Between the ages of 7 and 10 he had attacks of loss of consciousness four or five times a day; these attacks would be preceded by flushing of the face, and would last from several minutes to two hours;

during them he would curse and throw objects at those near him. Although he was ordinarily very bashful, he would, during these attacks, undress himself and run around the streets naked. He was apparently confused during these states but, up to a certain degree his actions could even then be influenced by shouting loudly at him. His mother stated that she noticed the peculiar skin condition on his neck as far back as his earliest childhood. At the age of 19 he was committed to the hospital for the insane in Nagykálló, where examination revealed the following positive findings: stigmata of degeneracy (low forehead, deformed ears, kyphosis), acanthosis nigricans limited to the sides of the neck, slow, shuffling gait, generalized hypertonia, exaggerated deep reflexes with abdominals absent on the right and diminished on the left, divergent strabismus, pale nerve heads especially on the temporal sides, slight secondary anemia, and idiocy. There were no evidences of neoplasm anywhere; ventriculography was also negative. All laboratory observations were negative. Treatment with thyroid had no effect.

The authors believe that the increased pigmentation, as well as the hypertrophy of the epidermis, is probably due to a functional disturbance of the endocrine glands, especially of the suprarenals; Frankenstein also attributes the disease to autointoxication from pluriglandular dysfunction and reports favorable results from the use of thyroid; Bolek's case was favorably influenced by the administration of suprarenal substance. (The evidences given by all these writers, including the authors of this paper, as to the endocrine genesis of the condition, while perhaps suggestive, is not at all convincing. Abstracter.)

KESCHNER, New York.

THE CELL AND ALBUMIN CONTENT OF THE CEREBROSPINAL FLUID IN CEREBRAL TUMOR. JENS SMITH, Deutsche Ztschr. f. d. Nervenh. **89**:278, 1926.

In 102 cases of cerebral tumor, only one third of the patients had a normal cell count, one-half had 2 or 3 cells, 25 per cent had more than 9, 15 per cent above 21 cells, and only 3 had more than 100 cells. The pleocytosis is commoner in tumor than in chronic encephalitis. There was on correlation betweeen type or location of the tumor and the cell count. An increase in albumin content was found oftener and was greater in tumor cases than in encephalitis.

ROY GRINKER.

BED-WETTING AND THE RÔLE OF DREAMS IN ITS CAUSATION. S. WEISSENBERG, Ztschr. f. Kinderh. **40**:343, 1925.

In Ukrania, during the War and the Revolution, fear of danger in many forms, warm drinks taken frequently to overcome hunger, and sleeping in very cold rooms, led to an increased amount of bed-wetting. The necessity of urinating would be postponed on account of the darkness, and unwillingness to arise in a freezing room out of a bed, which had become only slowly comfortable. Under these circumstances, the author knows by experience that there would then occur a dream of having arisen to urinate. In children the illusion would be perfect; they urinated in bed. Of all the causes of enuresis, Weissenberg considers the most important to be this provocative action of dreams, and suggests the use of camphor to diminish the depth of slumber and to soothe the irritability of the contractors of the bladder. Normal micturition follows the distention of the bladder, leading to a contraction of the detrusor, and a conscious relaxation of the antagonistic sphincter. In sleep, however, such relaxation does not occur unless a dream takes the place of

consciousness. Such relaxation occurs more readily in children who have been subjected to danger. The author had the opportunity of studying 112 subjects in an institution for the care of children who had been through many terrifying experiences.

EMERSON, Boston.

DEMONSTRATION OF HYPOPHYSEAL SECRETION IN HUMAN CEREBROSPINAL FLUID TAKEN FROM THE CISTERNA MAGNA. G. JANOSSY and B. HORVATH, Orvosi hetil. **69**:1033, 1925.

Before this, only animal experimentation proved that the secretion of the posterior lobe of the hypophyseal gland is always present in the cerebrospinal fluid. The authors used virginal uteri of rats for their experiments, and cerebrospinal fluid taken by cisterna or lumbar puncture. They found that the contraction of the uterus was always much stronger with cerebrospinal fluid of the cisterna puncture than that of the lumbar puncture.

Their conclusion is that the secretion of the posterior lobe of the hypophysis reaches the cerebrospinal fluid through the infundibulum, and is diluted and partly reabsorbed in the subarachnoidal space.

ZENTAY, St. Louis.

ON THE EFFECT OF LIPIODOL [IODIZED OIL] ON THE MENINGES. A. F. LINDBLOM, Acta radiol. **5**:129, 1926.

Subdural injections of relatively large quantities of an organic combination of iodine and oil of white poppy which, when injected into the bronchi does not seem to cause any irritation, when injected into rabbits gave rise to acute leptomeningitis, evidenced histologically by an extensive infiltration of cells (mainly leukocytes). It subsided in two or three weeks. In one case of seven it resulted in death. After the period mentioned no histologic changes remain, in spite of remaining large quantities of the drug. After intralumbar injection (5-10 cc.) in man, acute leptomeningitis develops with marked increase of cells in the lumbar fluid (exclusively lymphocytes). The meningeal symptoms have subsided within two weeks after the injection.

AUTHOR'S SUMMARY.

Diseases of the Ear, Nose and Throat

UNUSUAL CASE OF DEATH AFTER REMOVAL OF ADENOIDS AND TONSILS [HEMORRHAGE IN PONS]. H. BELL TAWSE, J. Laryng. & Otol. **41**:97, 1926.

The tonsils and adenoids were removed without special difficulty from a boy, aged 2 years and 8 months, under ethyl chloride anesthesia. The next morning, while sitting up to have his face washed, the boy fell back unconscious, and died within an hour. There was found a hemorrhage, the size of a large pea, in the center of the pons. All the other organs were normal.

[ARCH. PATH.]

PROGNOSIS OF MIDDLE EAR SUPPURATION IN CHILDREN. DOUGLAS MILLER, Tr. Edinburgh M. J. **33**:49 (March) 1926.

Complications of otitis media influence unfavorably the prognosis of diseases, especially among hospital children. Deafness results in a considerable number of cases, while intracranial infection is a potent factor in the mortality. Otitis is more common during the first two years of life. Although a frequent sequel of exanthematous diseases it is by far most often produced by the

common cold in the nose, and in infants under 6 months of age there may be in addition, bronchial catarrh which adds to the seriousness of the prognosis. Repetition of colds and the accompanying adenoid infection make it easy for infection to reach the ear.

While the symptoms of the acute condition are familiar, there are latent cases having indefinite or few symptoms referable to the ear. In such not uncommon attacks, the tympanum will be found lusterless and the cone of light absent, without redness or bulging. In this event incision is justified, and will result in escape of fluid and in the drop of the temperature to the normal.

Acute mastoid suppuration is frequent in streptococcic infection. The discharge from the ear stops suddenly, after seven to ten days, and swelling appears in the region of the mastoid, which if promptly operated on seldom passes into a chronic stage of suppuration. Neglect of treatment, repeated colds and the presence of adenoid growths permit the condition to become chronic.

The follow-up observations of 129 cases of acute suppuration occurring in the Royal Hospital for sick children during 1920 to 1923 was made in 1925. The acute duration of the otitis was in half of the cases under one week, the remainder lasting from two to four weeks. The incidence was more common in the first and second years of age. Of ninety-six patients traced, seven had died of causes not associated with aural disease. Deafness alone was found in 3 per cent, ear discharge in 5 per cent, and both in 6 per cent.

In eighty children with acute mastoiditis, occurring in the years 1920 to 1923, eight had intracranial complications and died. Of the seventy-two uncomplicated cases, thirty-nine were traced in 1925 and four were found to have died, meningitis, measles, tuberculosis and jaundice being the cause of death. Of the living patients, one was deaf, three had discharge, and two had both deafness and discharge.

The results in 207 chronic cases of suppurative otitis media were less favorable. In these, the treatment had been conservative. Adenoids were removed in seventy-five instances. In 143 patients followed up in 1925, 43 per cent had discharge, deafness, or both, two had died of meningitis and three of pneumonia.

In fifty-seven cases of chronic otitis, the treatment was by mastoid operation. Of these, forty-seven patients were followed up in 1925, and sixteen found cured, the others having deafness, discharge, or both, and two having died.

In twelve patients with tuberculous otitis media, eight had died, three had deafness or discharge, and one was free of these.

Miller emphasizes the importance of the type of operative procedure in mastoid disease. He regards the simple drainage of the antrum and air cells, without touching the middle ear as a more hopeful method of ridding the ear of infection and conserving the hearing. The removal of adenoids is beneficial. Radical operation is reserved for cases of complete destruction of the middle ear and for tuberculosis.

As prophylaxis, the infant should be protected against infection from colds, and nasal discharge is treated by instillations of 10 per cent mild silver protein in glycerine. Removal of adenoids, breathing exercises, and instruction as to blowing the nose are details needing attention in children.

In the discussion which followed the reading of Miller's paper, Dr. William Guy spoke of the etiologic menace of necrotic gingivitis, dental caries and oral sepsis. Dr. Logan Turner advised the appointment of otologists to care for children in hospitals, schools and child welfare centers. Dr. J. S. Fraser emphasized the need for early treatment and cure, and spoke of the greater

frequency among poor children. Dr. W. T. Gardiner has seen 400 cases complicating scarlet fever alone, and many occurring from measles and diphtheria. Removal of adenoids had cleaned up the discharge, as a rule, in an average of seven days. He advised removal of adenoids in every case of otitis media at the same time that paracentesis is performed. The nose or nasopharynx should be excluded as the cause of the otitis before a radical mastoid operation is performed.

NEFF, Kansas City, Mo.

CHRONIC NASAL DISCHARGE IN INFANTS. A. BROWN KELLY, Lancet **2**:1083 (Nov. 21) 1925.

There are three varieties of acute rhinitis from which a new-born infant may suffer. (*a*) Coryza neonatorum simplex, which resembles the ordinary head cold of the adult; (*b*) coryza neonatorum blenorrhagica, infection usually resulting from a leukorrheal or gonorrheal discharge in the mother's vagina at the time of birth; (*c*) coryza neonatorum syphilitica, which is a manifestation of secondary inherited syphilis. The differential diagnosis of these three types is discussed. Owing to the fact that children do not readily adopt mouth breathing, there is a tendency to suffocation when the nose is blocked. Sucking is also interfered with because the child must stop at frequent intervals to take a breath. Early and careful treatment of the symptoms and cause is most important. If nasal obstruction is marked a little epinephrine (1:10,000) may be painted in each nostril. Any crusts must be softened and removed. The catarrh is best treated by silver preparations. In the syphilitic subjects, a paint of colloidal mercury or an insufflation of calomel and sugar (1:25) may be employed.

Nasal suppuration is occasionally caused by an osteomyelitis of the superior maxilla. It is usually an empyema of a dental sac, the infection being hematogenous. This must not be mistaken for an antral empyema. A tooth projecting through the jaw should be removed, and thorough search made for sequestrums.

CHRONIC NASAL DISCHARGE AND SINUSITIS IN CHILDREN. A. BROWN KELLY, Lancet **2**:1132 (Nov. 28) 1925.

A foreign body may be the cause of a chronic nasal discharge, and if this is suspected in a particular case, great care must be exercised in manipulation. If the child is unmanageable it should be anesthetized. Before the anesthetic some epinephrine (1:10,000) may be sprayed in the nose to facilitate a more thorough examination.

Chronic rhinitis comprises two classes of cases: (1) When the lining membrane of the nose has undergone inflammatory thickening the more common causes being frequent colds, obstruction, septal deviations, etc.; (2) hypersecretion from the more general causes, tuberculosis, intestinal toxemia, nephritis, etc. Diseased tonsils and adenoids are frequently the cause of a chronic rhinitis and their removal will sometimes result in a cure of the nasal condition. If there is marked swelling of the inferior turbinates, the hypertrophy may persist after the operation and even require judicious removal with the scissors. The most intractable discharge is that which follows scarlet fever; it is benefited by mild cauterization with a solution of chromic acid (from 10 to 50 per cent). If there is any vasomotor disturbance the protein sensitization should be investigated. With reference to the general health and surroundings of the patient, a change is often followed by a cure. Sinusitis,

particularly of antral and ethmoidal origin may be present even in early infancy. They should be carefully investigated by roentgen ray, and if possible by exploratory lavage. The antrum can be reached through the inferior meatus in children over 8 years of age. The first in the treatment is the removal of the tonsils and adenoids. Instillations into the nose of 10 per cent argyrol are beneficial.

McNALLY, Montreal.

PHLEBITIS OF SINUS CAVERNOSUS. HANS BRUNNER, Monatschr. f. Ohrenh. **1**:2, 1926.

Detailed macroscopic and microscopic study of the pathology of affections of the sinus cavernosus as a complication of otitis media disclosed the unusually high frequency of this complication. Of twenty-two consecutive ear cases coming to necropsy, twelve had an involvement of the sinus cavernosus. Brunner's observations (54 per cent) are supported by Sternberg, the *prosecteur* of the Vienna Polyclinic. The highest percentage heretofore reported was 16 per cent (Uta).

Other intracranial complications associated with cavernous sinus phlebitis resulting from an otitis media were: thrombosis of sigmoidal sinus, two cases; meningitis, seven, and three with meningitis and thrombosis of the sigmoid sinus. In two instances, therefore, no meningeal symptoms were present, the infection extending as far as the cavernous sinus without meningeal involvement.

The route of extension to the cavernous sinus is (1) through the sinus petrosus, (2) retrograde transportation of thrombi from the sinus sigmoideus, and (3) through the veins of the plexus caroticus. The last mentioned route is important in the causation of cavernous sinus trouble, especially when the middle ear suppuration ulcerates through the eustachian tube into the pericarotic structures. In those cases in which the cavernous sinus phlebitis is accompanied by a meningitis, the mode of infection of the sinus is either through the venae cerebrales inferiores, or the carotic venous plexus, or through caries of the apex of the pyramidal bone.

In the cases studied, abnormal eye observations were infrequent.

GLOWACKI, Detroit.

MULTIPLE FOREIGN BODIES IN THE RESPIRATORY AND DIGESTIVE TRACT. GEORG RIEDEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:386 (April 27) 1926.

Multiple foreign bodies are mostly found in small children. Special care must be taken in the examination and extraction of fishbones. These are broken when they enter the trachea or the esophagus, and also when they are extracted. Therefore, meticulous care is indispensable in examining the operative field and during extraction. Riedel cites several cases of his own, and a number of cases reported by various authors.

AMBERG, Detroit.

THE INDICATIONS FOR ENTERING THE MASTOID PROCESS IN ACUTE MIDDLE-EAR SUPPURATION. OVE STRANDBERG and AAGE PLUM, Ugesk. f. Læger. **15**:369, 1926.

Symptoms warranting operation on the mastoid process in suppuration of the middle ear are discussed. The many various symptoms described are in general well known. The authors stress a daily observation of the patient and an operation as soon as symptoms appear, which is usually not before the fourth day.

SCHLUTZ, Minneapolis.

Diseases of the Skin

INFANTILE ACNE VULGARIS. SAMUEL AYRES, JR., Arch. Dermat. & Syph. **12**:12 (July) 1926.

Three cases are reported, the ages of the patients being 2, 5 and 22 months. All were boys. Only one other case report was found in a search of the literature of the last fifteen years.

HYPERGLYCEMIA. I. L. MCGLASSON, Arch. Dermat. & Syph. **13**:338 (March) 1926.

The author has made observations on the blood sugar in a number of dermatologic conditions, and has noted an etiologic and therapeutic relationship between hyperglycemia and certain of these conditions. Particular stress is placed on hyperglycemia in relation to infantile eczema, though the sugar determinations were made with blood from the mothers and not from the babies. In a series of 100 cases of infantile eczema, the reduction of carbohydrate in the diet of the baby or the mother resulted in great improvement or complete cure in all but one case. He mentions the use of a carbohydrate free diet.

THE USE OF MERCUROCHROME-220 SOLUBLE IN THE TREATMENT OF INFECTIOUS DISEASES OF THE SKIN. HUGH H. YOUNG, JUSTINA H. HILL and WALTER L. DENNY, Arch. Dermat. & Syph. **13**:465 (April) 1926.

As a result of the administration of mercurochrome, twenty of twenty-four erysipelas patients were cured or improved; ten of eleven patients with furuncles were cured and one improved; chancroidal ulceration in all of four patients healed rapidly; thirty of thirty-six patients with cellulitis and abscess were cured or greatly improved; in two cases of gas gangrene cure of the infection was effected; two of three patients with pemphigus were markedly improved without relapse, the third being improved but with relapse; of four cases of psoriasis two were apparently cured, one markedly improved and one improved but with relapse; of forty-four patients with leprosy, twenty-eight were improved with remarkable disappearance of skin ulcers.

JEANS, Iowa City.

FURTHER OBSERVATIONS UPON ACRODYNIA. A. E. VIPOND, Brit. J. Child. Dis. **23**:127 (April-June) 1926.

From his study of many cases of this disease the author concludes:

1. That it is merely a disease of the nervous system, and that a great part of the sensory system is involved.

2. That the children suffer agonizing pains in the arms and legs.

3. That this suffering produces an abnormal mental condition, which is manifested by a high degree of mental irritability and restlessness which is never noticeable in any other disease.

The ages of the patients are between 8 and 18 months. The duration of the disease is from four to six months. Recovery is complete, provided the patients recover, as they do in most instances.

The author found a small gram-positive diplococcus in every case of acrodynia that came under his care. Cultures were taken from the enlarged lymph nodes. Successful treatment was carried out with vaccine made from this diplococcus.

LANGMANN, New York.

DERMATITIS SCROFULOSA. BARBER and DOWLING, Proc. Roy. Soc. Med. **19**:33-34 (May) 1926.

The authors presented a group of cases under the designation of dermatitis scrofulosa. The eruption occurred on the limbs and trunk rather than on the face; when it did occur on the face, the patches had no predilection for the skin around the mouth. They were not fissured, unless irritated by soap and water, and they had a characteristic tint which varied from a pinkish-fawn color to a dirty brownish hue. The scales were larger and more flaky and fairly easily detached; the patches were not as a rule, circular, but were oval or irregular, and at times formed large sheets, particularly below the margin of the hair on the temples and on the limbs. The authors thought that dermatitis scrofulosa corresponded to the eczematoid trichophytid described by Jadassohn and Guth, and that it was an eczematoid reaction produced by sensitization of the epidermis to tuberculin. The reproduction of the eruption by the application of tuberculin favored this view. An interesting point was the rapidity with which the lesions disappeared under the influence of ultraviolet light.

In discussion Dr. Whitfield questioned the possibility of a tuberculous origin for this group, although he thought that eczema scrofulosorum of Boeck and his (Whitfield's) discoid eczema were identical.

Dr. Adamson also thought the eruption was independent of tuberculosis.

DISCUSSION ON THE ETIOLOGY AND TREATMENT OF INFANTILE ECZEMA. A. M. H. GRAY, Proc. Roy. Soc. Med. (Section of Dermatology with the Section for the Study of Disease in Children) **19**:73 (March) 1926.

The term "infantile eczema" is used to denote several types of superficial catarrh of the skin, but in this article only those varieties which attack the face were discussed. Facial eczema may be conveniently divided into three types.

1. The primary facial. 2. Those which begin on the scalp. 3. Those which occur in association with a general eczema. The author discusses the etiology at length. The main point in treatment is to stop friction. This can be accomplished by methods of restraint, also by keeping the child as far as possible in a room of equable temperature. An occlusive dressing, such as a face mask, is also of value in this respect. Zinc paste and the crude tar preparation are useful in the nonseptic cases to check the itching.

Reduction in the feedings is all that is necessary by way of dietetic treatment. Drugs that may be tried are antimony, thyroid extract and bromide, but none seem to give any definite improvement.

WILLIAMSON, New Orleans.

TWO CASES OF TRICHOPHYTOSIS DUE TO A FAVIFORM TRICHOPHYTON. E. BODIN, Bull. Soc. franç. de dermat. et syph. **32**:435, 1925.

Suppurating patches on a baby's face, and an impetigo-like lesion on a woman's wrist were found to be due to infection with a faviform trichophyton, probably *Trichophyton album*. In both cases the infection was apparently of animal origin.

[ARCH. DERMAT.]

CONSIDERATIONS ON SOME CASES OF ICHTHYOSIS. P. FORNARA, Clin. pediat. **8**:173-184, 1926.

Fornara reports four cases of congenital ichthyosis. They are of endocrine origin, mostly thyroid dysfunctions. The importance of syphilis in all these cases is stressed. It should be sought for very carefully and repeatedly, and,

in some cases, in spite of negative reports, antisyphilitic treatment should be instituted and given a fair trial. The treatment of the condition should, of course, be directed to the etiologic agent as far as it is possible to determine the cause. He advises also organotherapy with thyroid in every case. He treated one of his patients, in whom he obtained no results with syphilitic and organotherapy, with injections of foreign protein (milk) and reports a cure.

ZAMBARANO, Northville, Mich.

ETIOLOGY OF ERYTHEMA NODOSUM. E. FEER, Schweiz. med. Wchnschr. **56:** (July 17) 1926.

The author cites forty-five cases of erythema nodosum which he has observed, and his conclusions are as follows:

Although erythema nodosum is often accompanied by rheumatic symptoms, it bears no relationship whatsoever to acute articular rheumatism. He regards it as a specific disease of unknown origin, occasionally becoming contagious and appearing in epidemic form.

Genuine erythema nodosum attacks tuberculously infected persons exclusively. It therefore becomes an important indicator for existing tuberculosis in childhood.

The tuberculin reaction (Pirquet) is unusually violent — often vesicular or even gangrenous.

ELTERICH, JR., Pittsburgh.

THE QUESTION OF SEROLOGICAL RELATIONSHIP BETWEEN HERPES ZOSTER AND CHICKENPOX. E. LAUDA and E. SILBERSTEIN, Med. Klin. **22:**374, 1926.

In twelve cases of herpes zoster, the complement fixation test with blood serum of patients with varicella gave negative results. The authors do not draw the conclusion that this definitely speaks against the identity of herpes zoster and varicella in the sense of Bokay. They only emphasize the well-known fact that the etiology of herpes zoster is rather diversified, and only in rare instances is of varicellous origin.

ZENTAY, St. Louis.

Surgery and Orthopedics

SPLENIC DISEASES WITH SPECIAL REFERENCE TO SPLENECTOMY IN CHILDREN. I. RUSSEL KUHN, Arch. Pediat. **43:**407, 1926.

It may be said that the spleen, though its function is not at the present time fully understood, may best be considered as a filter placed in the blood stream under normal conditions to filter the red blood corpuscles which disintegrate daily. Under certain pathologic conditions, cells foreign to the circulation, such as nucleated red cells, myelocytes, bacteria and other toxic agents, are also filtered out, the retention of which adds greatly to the size of the organ. Splenectomy, generally speaking, is contraindicated in the leukemias and pernicious anemia. In Gaucher's disease and von Jaksch's anemia, removal of the spleen should be undertaken only after great deliberation. In many cases of Banti's disease, congenital family hemolytic jaundice and purpura hemorrhagica, splenectomy is definitely indicated. It is firmly established that the spleen is not necessary to life.

VON MEYSENBUG, New Orleans.

PNEUMOCEPHALUS. W. F. DANDY, Arch. Surg. **12:**949 (May) 1926.

The author's summary adequately covers the chief points in the article: 1. Pneumocephalus may follow any artificial communication between the para-

nasal or mastoid sinuses and the cranial chamber. It may also result from infections of gas producing organisms. 2. Sneezing, coughing, straining or swallowing is necessary to force the air through a bony and dural defect into the cranial chamber. 3. The intracranial air may be subdivided into four varieties according to location: (1) subarachnoid, (2) subdural, (3) intracerebral, (4) intraventricular. The intracerebral variety appears to be the most frequent. 4. The symptoms are mainly those of increased intracranial pressure, but rhinorrhea after sneezing or change of position is almost pathognomonic. A discharge of cerebrospinal fluid is usually present. 5. The absolute diagnosis is made by the roentgen ray. 6. Death results from infection and pressure in about 40 per cent of the cases.

N. Enzer.

Congenital Intermittent Luxation of the Patella. B. Formiggini, Clin. pediat. **8**:193-210, 1926.

Many of the luxations of the patella, formerly classified as traumatic, are now regarded as congenital. The luxation may be complete or incomplete; permanent or intermittent. Each of these varieties has its distinct symptomatology. In incomplete luxation the patella lies in front of the external condyle of the femur with the limb in extension. Flexion causes the patella to slip into its normal position. In the intermittent complete type, flexion of the leg causes the patella to be displaced toward the external condyle and its falls back into place on extension. In the complete and permanent variety the patella remains on the external condyle whether the leg is extended or flexed. Flexion increases the luxation. The anatomic predisposing causes for this condition are divided into three types by Chauvin and Liautard. The first type is characterized by the presence of a genu valgum. The second type has an arrested development of the external condyle, with or without hypertrophy of the internal condyle and a flattening of the trochlea. The third type is distinctive by the predominance of fibrous capsular lesions. A case in point is reported and the operative procedures discussed in detail. The simplest operations possible insure the best results. Entering the knee joint should be avoided.

Zambarano, Northville, Mich.

An Unusual Case of Foreign Body in the Vagina of a Two and One-Half Year Old Child. T. Stober, Monatschr. f. Kinderh. **32**:151, 1926.

In a girl, aged 2½ years, a vaginitis was found, due to foreign bodies introduced by herself and by her 6 year old brother. Under an anesthetic the following substances were removed: cherry stones, pieces of silica, bits of corn, eggshells, plum stones, and bits of wood.

Following removal of these foreign bodies, the vaginal secretion and associated cystitis stopped.

Acute Purulent Tuberculous Osteomyelitis. Richard Gralka, Monatschr. f. Kinderh. **32**:153, 1926.

This is the report of a case, in a child, aged 1½ years, with multiple osteomyelitis. The absence of elevation of temperature, as well as the lack of any other physical signs of an acute infection, suggested tuberculosis. On the other hand, the blood count was 17,000, with a high percentage of polymorphonuclears. The onset, too, was rather acute.

Tuberculin reactions were positive. To determine whether the infection was one of tuberculosis alone or a secondary infection of a tuberculous joint with pus organisms, a needle was introduced into one of the joints. The fluid obtained was a thick, whitish green pus. Microscopic examination, however, showed this pus to contain a pure culture of tubercle bacilli.

GERSTLEY, Chicago.

Miscellaneous

A PLEA FOR A STANDARDIZED METHOD OF ESTIMATING AND REPORTING HEMOGLOBIN VALUES. J. W. LINDSAY, E. C. RICE and M. A. SELINGER, J. Lab. & Clin. Med. **11**:737, 1926.

Few hemoglobinometers on the market are sufficiently accurate for clinical work. Hematologists should make a practice of reporting the hemoglobin content of the blood in grams per hundred cubic centimeters as well as per cent. From 14 to 17 Gm. per hundred cubic centimeters is suggested as the normal limits of the hemoglobin content of the blood, these figures being used in the same manner as the normal in blood chemistry. Wong's method of determining hemoglobin is sufficiently accurate for stadardization of hemoglobinometers in the clinical laboratory. The hemoglobinometer devised by Newcomer using a standardized glass color plate is recommended as the most accurate instrument for clinical work.

S. A. LEVINSON.

THE NORMAL INFANT AND CHILD. H. BROOKER MILLS, M. J. & Rec. **123**:99 (Jan. 20) 1926.

The average weight of the normal baby at birth is 7 pounds, and this should be doubled at six months and trebled at twelve months. All babies should be reared on human milk if possible. If not possible, cow's milk properly diluted should be given. Fruit juices should be started at the third month, weaning should begin at 9 months and the baby be completely off the breast at 1 year. The author is not in favor of waking the infant at regular intervals. A minimum interval between feedings should be decided on by the mother, and the baby will decide how much further apart the feedings may be. Plenty of water should be given between feedings.

Babies cry more from being overclothed than underclothed. Babies should be vaccinated at the fifth month, and between their sixth and ninth month should receive the three injections necessary to protect them from diphtheria and in the near future should receive the same treatment for the protection against scarlet fever.

Plenty of time for play should be allowed to the preschool and the school child. There is wonderful training for the powers of the growing child in play. Correction of the same faults in one child will be very different in another, depending on the temperament of the individual child. Control by love rather than by fear is the ideal method to employ. Do not compel children who are neither mentally nor physically fitted for such a strain to go through college. Young men and young women who select their own uninfluenced vocation in life do better than those who take up some kind of work because of parents' influence.

European School Hygiene and Other Notes. Edward A. Tracy, M. J. & Rec. **123**:284 (March) 1926.

The author took the privilege, while in Europe, of examining the school hygiene services of Frankfort, Vienna, Paris and London, paying special attention to the care given to the epileptic school child. In Frankfort for 50,000 school children they have fifteen school doctors, and three specialists. There are three schools for mentally backward children, and twenty-five beds are reserved in the University Psychiatry Clinic for chronic epilepsy and other mental diseases in school children. In Vienna for 150,000 school children there are seven full time doctors and thirty-five part time.

In Paris, G. Paul Boncous is in charge of the abnormal children, and by his efforts the diagnosis of early cases of epilepsy has progressed more than elsewhere in Europe. Special attention is given to the pretuberculous children.

In London, there are over one-half million of school children, with 2.7 epileptics in a thousand children.

There is a lovely home and school for epileptic children at Lingfield, about twenty-six miles from London. The National Hospital for Epileptics and the Maida Vale Hospital for the epileptic and crippled also receive school children.

Williamson, New Orleans.

The Value of Medical Inspection of Schools. E. R. Hayes, Nebr. State M. J. **11**:266, 1926.

In this report Hayes states that the number of defects have been reduced in ten years to 24 per cent of the 1,260 pupils in a small community. The following is a list of the defects found:

Hypertrophied tonsils, 128; defective teeth, 172; enlarged thyroid, 110; defective eyes, 34; skin lesions, 35; defective nose, 19; deformed chest, 21; abnormal heart, 16; defective hearing, 10; nutritional defects, 87.

Two per cent are substandard in intelligence; 9 per cent between ages of 6 to 17 years have enlarged thyroids, 96 per cent of these occurring in girls.

During the past year, the average attendance has not fallen below 85 per cent in any month.

Hamilton, Omaha.

Palpable Arterial Coats in Children. A. E. Vipond, Brit. J. Child. Dis. **23**:43 (Jan.-March) 1926.

The author has found palpable arteries and thickened coats in many children. The condition is a familial one and is nonsyphilitic in origin. Either the father or the mother will be found to have palpable arteries. The blood pressure in these children is normal, but a number of them are thin and undersized.

Statistics Concerning the Height, Weight and Other Measurements of Fourteen Hundred London Children. Bernard Myers, Brit. J. Child. Dis. **23**:87 (April-June) 1926.

The measurements used were the following: Head circumference, anteroposterior and transverse head measurements, chest circumference, circumference of abdomen, intercristal diameter, height, sitting height and weight.

The author uses the height-weight index in this study because, as he states, it is a simple method, but he cautions that this index must not be entirely relied on without a careful clinical examination. In each case it is important to note the general appearance and bearing of the child, activity, alertness,

movements of the limbs, etc.; past illnesses should be inquired into, and also the physique of the parents.

The author found that the sitting heights of American children were greater at 19 months, 2½ years and 5 years when comparisons were made, except in the case of girls at the last named period, when the clinic figures were higher.

At 13 years the females weigh more than the males in the English and American figures.

Sitting height increased 50 per cent from 2 to 12 years. The intercristal diameter increased between birth and 14 years, 161 per cent for males and 171 per cent for females.

LANGMANN, New York.

POSTMORTEM CAESAREAN SECTION IN TWIN PREGNANCY, WITH SURVIVAL OF ONE CHILD. GEORGE W. YULE, Edinburgh M. J., Obst. Soc. **33**:49, 1926.

Yule reports one living twin delivered by cesarean section, fully five minutes after the mother had been judged to be dead. The infant required resuscitation for half an hour, but lived and left the hospital on the tenth day in good condition.

NEFF, Kansas City, Mo.

CASE OF TRANSPOSITION OF VISCERA. BERNARD MYERS, Proc. Roy. Soc. Med. (Clinical Section) **19**:2 (Jan.) 1926.

The author reports a case of complete transposition of the viscera in a boy, aged 6 years and 3 months. The patient also showed double talipes equinovarus. His cheeks, nose, ears and extremities were of a dark slate color. The other parts of body were only slightly cyanosed. The fingers and toes showed advanced clubbing. A systolic murmur was heard over whole precordium. The electrocardiogram showed inversion of PRT in lead I. Blood count: Hemoglobin, 147 per cent; color index, 0.7; red blood count, 9,960,000; white blood count, 8,600. There were no abnormal cells, and the Wassermann reaction was negative.

CASE OF CONGENITAL CYANOSIS. BERNARD MYERS, Proc. Roy. Soc. Med. (Clinical Section) **19**:3 (Jan.) 1926.

A girl, aged 5 years and 10 months was admitted to hospital because of "backward" mentality and of "blue color." The blue coloration was first noticed when she was 6 months old. She had never spoken and had always been subject to "fits of restlessness." No history of attacks of extreme cyanosis could be elicited. Blood count: hemoglobin, 65 per cent; color index, 0.9; red blood count, 9,100,000; white blood count, 8,125. On spectrum analysis, oxyhemoglobin was seen to be present. Complete aphasia was present. The child understood some things which were said. Cyanosis of whole body, and clubbing of the fingers were present. Examination of the heart was negative. In cyanosis, we think of causes which are due to lung conditions, and to those due to some error of the heart and blood channels. Erythemia and enterogenous cyanosis have also to be borne in mind. The appearance of cyanosis during infancy and its continuous presence with marked clubbing, absence of splenic enlargement, the high color index, absence of albuminuria, headache, or dyspnea, would rule out erythemia. We can eliminate enterogenous cyanosis because of absence of meth-hemoglobin and sulph-hemoglobin. There is no lung affection, we can rule out the pulmonary causes of cyanosis. Absence of murmur rules out pulmonary stenosis, patent ductus arteriosus, etc. The

final choice lies between a trilocular heart and transposition of the great vessels leaving the heart, or to a junction between aorta and pulmonary arteries. The most probable cause is transposition of great vessels. The roentgen ray gives no help.

A Case of Symmetrical Synovitis of Knees. W. R. Briston, Proc. Roy. Soc. Med., Section of Orthopedics **19**:31 (May) 1926.

This condition was reported in a girl, aged 6 years, whose Wassermann reaction and Sachs-Georgi flocculation test were both negative; the blood count was normal. The urine was normal, a gonococcal complement deviation test on the blood and joint fluid were negative. The fluid of the joints showed a preponderance of polymorphonuclear cells. Cultivation of 2 cc. of fluid yielded one colony of *Staphylococcus aureus*. One cubic centimeter of joint fluid was injected into the peritoneum of a guinea-pig. Three weeks later the animal was killed, and no sign of disease was found. Cultures of the peritoneal cavity were sterile. Pirquet's test was negative. The lesions were evidently due to a general infective process of which the exact cause remains "not proven."

Congenital Stenosis of the Esophagus. C. C. Beatty, Proc. Roy. Soc. Med. (Section for the Study of Disease in Children) **19**:38 (March) 1926.

The patient, a girl, aged 3 years, commenced to vomit at the age of 12 months, when she was weaned. Vomiting occurred immediately after a few mouthfuls of solid food had been taken. Roentgen-ray examination showed a marked narrowing of the esophagus at the junction of the lower and middle third. Bougies were passed up to a diameter of one third of an inch, but there was no improvement in swallowing.

Williamson, New Orleans.

The Eosinophile Blood Picture in the Japanese Infant, with Special Reference to Exudative Diathesis. T. Tomita, Monatschr. f. Kinderh. **32**:97, 1926.

The author made blood examinations of 1,032 Japanese children to compare them with the findings of the average European child. In this series, 845 were under 1 year; 162 under 2 years; fifteen under 3 and 10 under four years. Five hundred and ninety-nine were boys and 433 were girls. Practically all of them, that is 98 per cent, were breast fed.

The findings were much the same as in European children. In exudative diathesis there is a slight increase of the eosinophils, but this is not absolutely constant and in some cases there may even be a slight diminution. Anemia causes little change in the blood smear. Children with anemia plus exudative diathesis show little change from the normal. In a general way, the relation of lymphocyte to polymorphonuclear is the same as in Europeans, but it is always quite variable and somewhat inconstant.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, April 13, 1926

GEORGE E. BAXTER, M.D., *President, in the Chair*

ALBUMINURIA IN CHILDREN. Presented by DRS. J. K. CALVIN, BERTHA ISAACS and JACOB MEYER.

In an attempt to determine the possible causes of nonnephritic albuminurias, which at present are classified in a confused group of benign, physiologic, growth or orthostatic albuminurias, two groups of children in largely different environmental and physical conditions were analyzed. One of the groups studied was composed of children having no special complaints or illnesses. They were recruited from the homes of the very poor in which hygienic conditions were at their worst, diets were improperly balanced, and teeth, tonsils and other potential foci of infection were neglected. Consequently undernourishment and anemia were common. About 200 of these children were examined, the sexes being about equally divided and the average age 8 years. Approximately 70 per cent were underweight; 63 per cent had some obvious foci of infection, especially infected tonsils and adenoids, nasal sinusitis or abscessed teeth. Albuminuria varied from a trace to ++, 45 per cent of the boys showing albumin and 70 per cent of the girls, or 60 per cent of the total number of children examined. The microscopic findings were abnormal in less than 1 per cent of the cases. Eighty per cent of the total albuminurias (60 per cent) showed evidence of malnutrition and foci of infection.

The second group studied consisted of children living in a well regulated orphan's home under the supervision of a watchful physician. Their hygienic surroundings were very good. They lived a well regulated life with the proper balance between school work, play, recreation, and rest. The meals were properly balanced, foci of infection were eliminated as soon as discovered, and periodic physical examinations were made. About 350 of these children were examined, the sexes being about equally divided and the average age being 11 years.

In this group, 10 per cent of the boys and 20 per cent of the girls showed albuminuria. Most of the albuminurias were transitory in character, disappearing and reappearing again over months of time. Morning and evening specimens from these subjects showed that most of them were not orthostatic in character, only 3 per cent of the children examined and 22 per cent of those showing albuminuria were of the orthostatic type.

As a result of the studies, these investigators believe that some of these albuminurias may be considered physiologic or growth albuminurias, but that many other etiologic factors may be present. Foci of infection, especially chronically infected tonsils, adenoids, nasal sinusitis and abscessed teeth, are the chief offenders, the removal of which often results in the permanent disappearance of the albuminuria. Undernourishment and anemia often are associated with albuminuria which respond to the overcoming of these factors. We do not believe that these factors (foci, anemia, etc.) have any direct specific effect on the kidney. They probably are a part of the picture associated with

malnutrition or an under par condition with faulty blood volume and blood flow, etc., resulting in hypofunctioning or impaired functioning of the body tissues. In short, the albuminuria is merely one symptom of a general systemic disturbance in these cases.

The following classification for the convenience of the physician are offered. Benign albuminuria may be divided into three groups:

(*a*) Malnutrition albuminuria; frequently associated with anemia, underweight, and systolic basal murmur. Foci of infection, especially infected tonsils, adenoids, nasal sinuses and carious teeth are common causes of this malnourished condition.

(*b*) Orthostatic albuminuria, associated with posture.

(*c*) Developmental idiopathic or "growth" albuminuria; including the terms juvenile, puberty, cyclic, transitory, intermittent.

According to this experience, the simplest methods of treatment should be followed. The authors recommend first, an intelligent explanation of the nature of the findings to the parent, correction and elimination of apparent foci of infection, improvement of nutrition, and treatment of anemia. They do not advise any excessive rest cure or restricted low protein diet as a remedy. Such measures may actually result in harm to children who are physically under par. The child should be allowed to lead a normal life, with only such restrictions as are necessary to overcome any malnutrition; otherwise freedom in every regard has proved to be the best remedy. Special emphasis should be laid on the physical development of the orthostatic group.

THE TREATMENT OF NEPHROSIS. Presented by DR. C. A. ALDRICH.

This paper appeared in full in this journal, August, 1926.

DISCUSSION

DR. JOSEPH BRENNEMANN expressed his appreciation of Dr. Calvin's paper, and especially of the sanity of his conclusions. He thought anyone whose medical memory went back twenty years or more must be impressed by the change in our attitude toward albuminuria then and now. It was formerly thought that albuminuria was a very serious condition, and it was interesting to find that so large a number of apparently healthy children have a definite albuminuria at one time or another.

In discussing Dr. Aldrich's paper, he emphasized the method of study that was pursued. When Dr. Aldrich came to the Children's Hospital five years ago, Dr. Brennemann suggested to him that it might be well to study nephritis intensively, as it was a limited and very hazy field. Some 150 cases have been studied in that time. The sphere of the work was much broadened when Dr. McClure suggested that it might be possible to tell something about edema by injecting physiologic sodium chloride solution under the skin, and timing its disappearance. He was sure all would agree that some very interesting work had come from that. In his opinion, Dr. Aldrich's paper presented the broadest minded attitude, and the sanest conclusions of any paper that he had seen on this subject. After hearing of the work in St. Louis on drainage of the nasal sinuses, he was disappointed in reading Clausen's paper to find that, of his eleven patients, not more than one or two showed unmistakably that the cure in the case came from drainage of the nasal sinuses only, and not possibly from a mere cessation of an infection. It was not of much value to conclude that because edema disappears after drainage of the nasal sinuses one has cured the patient, unless one follows the cases for a number of years as Dr. Aldrich

had done. He did not believe the impression gained from the literature was that one could have nineteen or twenty periods of edema in seven cases. It seemed to him the only logical conclusion was that nephrosis in all probability is due to an infection, possibly with the staphylococcus, although this had not yet been demonstrated. In one of Dr. Aldrich's cases cure occurred following drainage of a streptococcal infection about the eye. The infection is practically always nasal or paranasal, and it has been demonstrated by many that following drainage of the paranasal sinuses cure occurs, but in only seven of Dr. Aldrich's nineteen cases of edema did the result follow the opening of an abscess or the drainage of a sinus. In all the other cases the children had an upper respiratory tract infection, and the edema disappeared after the infection cleared up.

Dr. Brennemann believed all that could be said at the present time was that nephrosis was due to an infection, and that when this was cleared up the patient would be free from edema. The commonest infection in childhood is a nasal and throat infection, hence nephrosis occurs more often with such infections. The result follows more evidently and more spectacularly when a sinus is drained, but in the great majority of Dr. Aldrich's cases it followed the termination of an infection without sinus drainage. It followed more slowly, naturally, but just as logically. There seems to be a constitutional predisposition to nephrosis, which might account for the fact that only certain subjects develop such attacks.

Dr. J. H. Hess congratulated Dr. Calvin and his co-workers on the practical value of their work. He believes they have accomplished much toward relieving the minds of the profession of the idea that the existence of albuminuria always meant something serious. Dr. Hess concurred in everything Dr. Brennemann had said regarding Dr. Aldrich's paper.

Dr. George E. Baxter asked Dr. Calvin whether there was any possibility of contamination from vaginal discharge in the girls, and whether the urine was taken in the ordinary manner or by catheters.

Dr. Calvin replied that the urine was taken by the ordinary method, and in only a very small percentage of the cases were there any abnormal microscopic findings. The girls were all examined for vaginal discharge, and he felt that there was little likelihood of contamination from that source.

Dr. Jacob Meyer (closing): One rather hesitated to speak of albuminuria as a benign disorder. Physiologists do not accept it as a physiologic phenomenon. Although it is well known that to a certain extent albumin may be found in normal urine by the most refined technic, this is probably the criticism to which the test is subject. Too great care and refinement may cause a great deal of worry. He thought the conception of albuminuria fitted in with some of the new views, which had been so well expressed in an editorial in the *Journal of the American Medical Association,* in that changes in the blood flow are supposed to account for the albuminuria. In nephrosis the albumin comes from the tubules, and if one has a conception of the temporary phenomena associated with malnutrition, one can readily conceive of such a clinical phenomenon. They have been careful about using the term "growth" or developmental albuminuria, and have stressed the point that orthostatic albuminurias are very infrequent as compared with the statements of other workers. They believe that there is a real developmental albuminuria. They are following a number of children and their weight and height curves in association with albuminurias, in an effort to estimate the relationship between these conditions

in trying to prove a developmental albuminuria. They have been impressed with the fact that girls present a higher percentage of albuminuria than boys, and think this is accounted for by the fact that the height and weight disturbances are more marked in girls than in boys.

DR. ALDRICH expressed his great interest in Dr. Calvin's paper, and considered the work of particular value to the profession.

AMERICAN PEDIATRIC SOCIETY

Thirty-Eighth Annual Meeting, held at Niagara Falls, Canada, May-June, 1926

J. H. MASON KNOX, M.D., *in the Chair*

FOURTH SESSION

THE CONTRIBUTION OF PSYCHIATRY TO PEDIATRICS. Presented by DR. HERMAN M. ADLER (by invitation).

Pediatrics confines itself obviously to children. Psychiatry, on the other hand, deals with mental disorders of all human beings. It will be seen, therefore, that pediatrics and psychiatry overlap. There is need for a definition or a delimitation of these two fields. While pediatrics is a specialty, it is based on the same logic and the same body of scientific evidence as general medicine. This is not quite the case with psychiatry. The early attempt to convert psychiatry into a specialty, which like other branches of medicine should base its diagnosis on organic changes, predict a course and end-results, and thereby a treatment, has not yet been achieved; there have developed, however, other approaches to the human mind.

There is another side to the problem, and that has to do with the behavior of the organism as a whole. There are some behavioristic phenomena, of course, which are of importance to the pediatrician, such as those accompanying high temperature or delirium, but he does not focus his attention on these phenomena, he merely utilizes them in obtaining his diagnosis. The psychologist and psychiatrist are largely interested in the behavior itself and the mental causes behind it. Both of these fields have been widened to include many conditions which are on the borderline of pathology and which sometimes extend over into the field of health. All pathologic conditions have a direct and an indirect effect on the central nervous system. The direct effects, such as paralyses, deafness, lameness, deformities and weaknesses, are the obvious problem of the pediatrician. But the indirect effects, such as the child's emotional reaction to his inability to do things, or to the mortification at his abnormal appearance, or the habits set up by uncontrollable bursts of exacerbation, producing an unfriendly attitude of others toward the patient, and this in turn producing more violent reactions on his part — a very common situation — are not so definitely his problems. These problems, often the most important from the point of view of the child's whole future happiness, are sometimes entirely overlooked. Later, the parents deal with them as "moral" problems, but to the psychiatrist who sees the end-results, it seems that more could be done if the pediatrician had a greater appreciation of these dangers and were better able to instruct the parents in the early care and training of

these children. Outstanding examples of physical factors causing mental problems are anemia, malnutrition and endocrine disturbances.

A better appreciation of the child, as a whole, is necessary on the part of the psychiatrist and the pediatrician alike. The psychiatrist, in dealing with children, must depend on pediatrics. The pediatrician, on the other hand, will require the psychiatrist in an increasing degree when understanding in a given case is required beyond the immediate organic problem. This condition must be recognized by the medical schools, and provision made in the curriculum of the graduate schools to give the pediatrician courses bearing on psychiatry in order to practice his specialty, and correspondingly the psychiatrist of the future must be acquainted with the processes of pediatrics if he wishes to deal with the problems of children. As our knowledge becomes more accurate and complete, we shall experience the same tendency toward synthesis which is now manifested in the fundamental sciences, such as physics and chemistry. When this point is reached, the dividing line between pediatrics and psychiatry will largely disappear.

CAN THE PROBLEM BE APPROACHED FROM ANY OTHER POINT OF VIEW? Presented by DR. BRONSON CROTHERS, Boston.

We as pediatricians, being the only specialists who claim to preserve the virtues of the general practitioner, must take serious notice of the invasion of our field by others. Unless we are willing to confine our interest to the bodily functions, we cannot avoid consideration of intellectual and emotional manifestations. The moment we fix our attention on behavior as opposed to structure, we must necessarily begin to use "psychologic" or "psychiatric" methods. The only question that then arises is whether we use the best methods available.

The psychiatrists and the psychologists have been generous, even lavish, in offering help and advice. The more or less bewildered pediatricians need to pause a bit before so many riches. We can, I believe, make a wise and profitable selection only if we study the display with considerable care. In the first place, it is by no means certain that the most elaborate and most accurate tools will be best adapted to our needs. As general practitioners, it is our chosen fate to be perpetually selecting bits of technic, from farmers, from chemists and from experts generally. We have, on the whole, done creditable work on nutritional disturbances, the control of infection, and so on.

I believe that the next logical step is to borrow all possible efficient tools from the mental experts. It is very tempting to start with highly intricate devices like the psychometric tests and the analytic vocabulary. I believe, however, that a less dramatic but more effective method is to start with the less advertised virtues of interest and patients; after all, these are the fundamental assets of psychiatry.

With curiosity well aroused, it is extraordinary to see how many problems can be recognized. With patience, many can be solved. As time goes on, bits of technic can be added, occasional names can be attached to syndromes and gradually a method, useful to us and to our patients, can be worked out.

We as pediatricians cannot possibly be expected to become sufficiently familiar with adult psychiatry to use all the technical psychiatric methods, but we have the great advantage of being an important part of the educational machinery. As I see it, if we do not take an industrious interest in education, in its broadest sense, we will have to be prepared to hand over one of the most fascinating parts of pediatrics to psychiatry. This sacrifice seems to me unnecessary.

The obvious ideal is close cooperation with psychiatrists, with increasingly effective work all around.

DISCUSSION

DR. HAYNES: When a jury is chosen — and those who discuss a paper are in a certain sense a jury — it is certain that those who do not read the newspapers and do not know much about the reported facts are chosen. I wonder when Dr. Carpenter asked me to discuss these papers whether he had that in mind. But it is a great privilege to have been invited to take part in this discussion, and to have heard from slightly different points of view two such sane and illuminating papers.

Dr. Adler has pointed out the importance of physical defects on the mental life of the child. I think likewise that we who are practicing medicine realize more and more how important is the influence of the mental processes on the health of the child. The illness with which he is battling is often removed when a wrong mental attitude toward life is changed for a correct one. From the side of pediatrics the mental life is important, and from the psychiatric standpoint the physical condition is important. As Dr. Crothers has said, it takes a great deal of time, patience and interest to make a mental examination and to straighten out mental difficulties.

One thing I heartily concur in is the attitude of the pediatrician toward himself, and I should like to emphasize that I think we want to get away from the idea that we are specialists. For our own part, we do not have that idea, but others have it and it should be corrected. We are not specialists; we are general practitioners. We have an age limit to our practice that naturally gives us the problems of infancy, growth and nutrition. These are special problems, but we are not specialists in the sense that the otologist and the laryngologist are. I once had a very amusing experience. I was called to see a child because I was a "weaning specialist." That is getting it down to a fine point. When the pediatrician has some knowledge of the ear, he is better able to select an ear specialist. The pediatrician who knows enough about the ear often does a myringotomy, but the point is that he is better able to choose a specialist by knowing something about the specialty. So in psychiatry, when he knows something in that line, he knows when to bring in a specialist in the region of intelligence and behavior. Dr. Crothers is correct in saying that we should learn something about psychiatry. Here we shall not become as capable of making a mental examination as the skilful psychiatrist, but the pediatrician has the advantage of knowing the parents and the child from infancy. In this he has a chronologic advantage over the psychiatrist called in for mental or behavioristic abnormality. The pediatrician, if he calls in the psychiatrist, should not remove himself from the picture.

What Dr. Crothers has said about the importance of psychiatry is correct, and one might suspect from what we have heard that psychiatry is learning. As a science, psychiatry is not of long duration and is in the position that in ten years the opinions of today will be changed. It is important to know it, however, and to remember that it is a human mind judging the problems of another human mind and is dependent on personality. We choose a surgeon not only for his skill but for his character and his honesty, and because he is a man with whom we are willing to trust our lives.

With Dr. Crothers, I think we may become skilful enough to handle psychiatric cases in children, but I do not think the psychiatrists will ever have enough men to handle all the work to be done. In the management of psychiatric cases, there is danger from unqualified workers. The truth in the words stated may be the truth to the person who has spoken them, but if

they do not bring to the hearer the same meaning that the speaker intended to express it is not true to him. A great deal of talk on the part of psychiatrists has brought fears that children were abnormal mentally when perhaps they were not, owing to the fact that the psychiatrist has not made himself understood.

It is advisable that we shall learn something of psychiatry so that it may not fall into the hand of unqualified persons. We have had that situation before, when we neglected the underlying principles of osteopathy. We did not use it. We did not investigate it. If we had done this in the beginning, perhaps we would not have osteopathy today. We had the same situation in reference to malnutrition. Everyone rushed in where angels feared to tread, and there were nutrition workers all over the country. Here is a question that touches so many homes that the same danger may creep in. The psychiatrist should do all he can to prevent such an unfortunate occurrence. With things as they are, for a time we may have to go along with the truth and God's own common sense, which is more than knowledge.

DR. H. B. WILCOX: For one limiting his practice to infants and children, a discussion on psychiatry seems at first thought far from his experience and ability. On the other hand, the pediatrician has something to do with this branch of medicine in most of his daily work. It would be, perhaps, possible for him to prove his right to be considered a superpsychiatrist, because everything he does must stand the acid test of parents, grandparents and friends. However concretely anatomic the problem may be as presented by his patient, the pediatrician's task as applied to the family is often pure psychiatry.

Dr. Adler has spoken of the relatively short time spent on his patient by the pediatrician, and says that we may very well see fifteen children in an afternoon, while the psychiatrist spends days studying one case. Were Dr. Adler as much of a pediatrician as every pediatrician is a psychiatrist, he would have avoided this erroneous idea.

The pediatrician must treat the mind and body of his young patients and, at the same time, the minds of his parents. He is pediatrician and psychiatrist, or he fails. Indeed, the nature of our subject makes this inevitable. We are dealing with an age in which rapid change — growth and development — make disharmonies, temperamentally and physically, most common. Consider the task confronting every child at birth, the necessity for compliance with the normal rate of increase in height, weight and bulk, the acquirement of physical and psychical coordination, the development of his special senses and the necessity of doing all this symmetrically. Truly, the idea of occupation and occupational disease can be applied to pediatrics. Every child is faced with one occupation in common, that is, that of growth and development, and this occupation controls his first year and largely influences his first ten years. The occupational diseases would then be considered deviations from normal progress in growth and development. If one considers the enormous importance of the stage of development, the rate of growth and the state of nutrition on the reaction of every child to his normal environment or to his expression of morbid states, it is easy to understand the peculiarities shown by these young patients when both well and sick.

Jacoby has said that at no period is the body so liable to disharmonies of structure or function as when in a state of rapid growth, and change from immaturity to maturity. This is true and finds its application in prenatal and postnatal periods.

We learn from the embryologist and anatomist that normal prenatal development is continuous. When interfered with, either structural or functional

abnormalities are likely to develop. We learn that there are periods in prenatal life of rapid growth, and that if interruption occurs in these periods, monsters may result. We learn that there are periods of slow growth and that, if interruptions occur in these periods, abnormalities are less likely to result. The application of these facts to the pediatrician's responsibilities is evident.

In postnatal life the same point may be made, that is, the influence of rate of growth, stage of development and, of course, state of nutrition on both structure and function. This is illustrated by certain phases of rickets. It has doubtless been the experience of everyone to see rickets develop in spite of proper nutritional, hygienic and antirachitic treatment. When this happens, it is most likely to occur at the period of most rapid growth. Thus, the pediatrician seems to be dealing always with that period of life most susceptible to disharmonies of structure or function. That part of the young organism showing most rapid growth is the central nervous system, increasing as it does many times more rapidly than any other group of organs in the body. The young human animal is therefore changing rapidly mentally and temperamentally as well as physically.

These facts give to pediatrics its right to exist as a specialty and perhaps also justify the belief that psychiatry is of necessity closely allied to the pediatrician's task.

DR. KERLEY: I agree with all that Drs. Adler, Crothers and Wilcox have said. The child's development, education and the way in which mental problems should be handled should be taken under consideration, but not all parents have pediatricians and psychiatrists to point out the defects in their children.

Mental and other defects must be discovered early in the preschool age, before the child comes under the observation of the school physician.

A great mass of defective children are never seen by trained observers. There is only one way in which this can be accomplished, and that is by state supervision of all children. By this means only will mental defectives and the physically unfortunate be discovered. An auxiliary to state health departments must be established for the purpose of visiting the child in its home, and advise parents as to proper procedures; in this way only will the child be brought to the attention of those particularly skilled in its management.

DR. LUCAS: Almost fifteen years ago, Dr. Southard said that in no field of medicine was it more important that psychiatry should be understood than in pediatrics. He said that was the beginning of all psychiatry and psychiatric work. I think most pediatricians would be better pediatricians if they were acquainted with psychiatry; this also applies to psychiatry, the psychiatrist would be better if he knew more about pediatrics. I do not think there is a greater truism than that of the Jesuits, who said that if they had complete control of a child until he was 7 years old, they could let him run and not fear. Few psychiatrists see the child before the age of 7. The work is started in the juvenile courts with adolescents, and only now are they gradually going back to the earlier years. Dr. Southard said that habits began in the nursery; there you saw the first signs of temper and fear, the beginning of bad food habits and why there were certain mental slants. The pediatrician has an advantage over the psychiatrist because he knows the family; he need not analyze family complexes because he knows the reactions of the father and mother, and their effect on the child. I agree with Dr. Crothers when he says we should study some of the technic of the psychiatrist. It is not a lack of interest, but we do not have time to analyze and establish the cause of the

psychiatric difficulty. The speakers are correct when they say that psychiatry is of the greatest assistance to us, but we enter the field on the preventive side. We should be prepared to prevent these special complexes in their incipiency before they require a psychiatrist. I do not think it is wise to attempt psychiatric work without the assistance of the grandmother and aunt. Through them we learn how the nursery is managed. If the nurse or governess is filling the child with fear, she is just as much a menace as though she had tuberculosis. I have seen complexes in a child just as hard to eradicate as tuberculosis, and there are just as many, perhaps more, nurses and governesses producing these complexes as have active tuberculosis. Failure on the part of the pediatrician to discover these facts early means that the child is headed for a complex. You can prevent this if the condition is discovered early, and this is the field in which the unconscious psychiatry of the pediatrician may be effective. When the problem requires hours to make an analysis and work out the treatment, the aid of the psychiatrist must be sought.

DR. FOOTE: I am somewhat disappointed, as I thought we would have a little more excitement, but so far those from whom we might have expected controversy are in accord. Dr. Adler handed over to the pediatrician many problems which the pediatrician would prefer to give to the psychiatrist. Many of the speakers have struck the vital matter when they say that the main purpose, so far as we are concerned, is the education of the mothers with whom we come in contact. As a matter of fact, there is now a marked national movement for mental hygiene, and there are many habit clinics in larger centers, yet we must realize that this movement, so far as mental hygiene is concerned, is in the same condition as to progress as the movement for child hygiene was twenty years ago. This is purely a matter of public education. Until the mass of people are educated we will not have results from mental hygiene. One reason is that our knowledge is not as exact as it might be. There is a certain zeal in the fields of psychiatry and neurology similar to that found in the realms of theology and philosophy, which makes it difficult for one not well balanced to steer a proper course. As a matter of fact, there are today two schools of child psychology, the leaders of which are publicly debating and denouncing each others' theories. Naturally, it is difficult for the public to know what to believe, or to know what one or the other of these eminent professors believes. The pragmatic school got rid of the concept of the soul a long time ago, and substituted for it the mind. But the behaviorists find even the concept of the mind a needless encumberance. Dr. Cattell, in speaking of the situation said: "Psychology long ago lost its soul, but I understand that now it is losing its mind." When professors disagree, who shall decide? Certainly not the bewildered public.

Mental hygiene is most important, and if people can be educated to it it will save psychiatrists more trouble than any other measure that we have. But we must wait and weigh our facts, stop arguing over theories and get down to essentials.

What is knowledge today becomes ridiculous tomorrow. We are groping in the dark — let us not be too proud of our knowledge. If we can assemble out of the heat of controversy a few demonstrable facts, and place these in the hands of all who talk to parents — the pediatrician in child welfare centers as well as the pediatrician in his office — we will make some progress. Mental hygiene is not for children; it is for parents primarily. Once let an ideal program be made possible, and the community will have no problem but that which the psychiatrist will be obliged to handle.

DR. KOPLIK: It seems to me that what we need more than anything else is to realize the vast number of cases in existence, and how difficult it is to draw the line between the patients needing treatment and those who can be considered normally peculiar. What we need is the confidence of the public. There are so many different opinions given by physicians, that the public is driven from pillar to post to get a child treated properly. It requires a great deal of time on the part of everybody in order to treat a child who is abnormal. I make bold to state that few pediatricians have given time to the study of the mind, not orthopedic deformities, birth deformities, etc., but to the mind, as Dr. Adler has pointed out. I have no hesitation in acknowledging my complete ignorance of pure psychologic studies. I have several cases in mind in which I placed too much confidence in the family, as, for example, in a case following encephalitis the boy was treated by the family by a method not advised by the physician. When I have to diagnose the case of a peculiar child, I am very much at sea, and I would gladly turn it over to some person who can understand the mind of the child.

We have not educated the public, and the family does not follow the advice and directions of the physician. We are too much on the lookout for some method which will accomplish results immediately. I have now such a case under observation, and a psychiatrist also in observing the same case. I saw a boy, aged 11 years, who was very bright at school, and has the development of a boy of 15, but he is very peculiar mentally. You might as well tell his parents that you do not want them to come into your office any more, as to tell them to go to a psychiatrist, for the boy is bright and they do not think that there is much the matter with him. In other words, we ourselves are to blame for these conditions and the failures. If we do not know a thing, we should say so. We owe it to the public not to let them go from one clinic to another. The number of children who are peculiar is enormous. The problem is too deep for immediate solution; we have only begun to scrape the varnish off the real problem.

DR. ADLER: I want to express my appreciation for the opportunity of not only being here but of hearing the discussion. It has been a better discussion than I have heard in many psychiatric meetings.

There are one or two points on which there is some misunderstanding. I am in entire agreement with the main point Dr. Crothers made. Dr. Crothers said something about the normal child, and that the pediatrician has the opportunity of seeing this child and therefore of preventing a great deal of difficulty in the future. I feel that any medical study depends for progress not on the normal subject but on the pathologic subject. It is necessary not to minimize the importance of deviations from health.

In the second place, with reference to how much the pediatrician should do in psychiatry, there is no bar to everybody becoming a psychiatrist except the time it takes to become acquainted with the subject. Human behavior is the special subject of the psychiatrist, but also of pediatricians, sociologists and various other investigators, and from whatever angle we approach it, the study of human behavior takes time. We cannot transfer our ideas from any medical specialty to juvenile behavior without patient study. It has been my experience that when we get even a new psychiatrist into this work he finds he has had no training for it. Unless he has been working in this particular field, it takes him at least three months to find out what it is all about. It may look like charlatanry to some, because we have to take note of such subtle changes and slight deviations that it takes time to accustom the eye to detect

these and to get information by simple questions and paying attention to special problems. I agree with many of the pediatricians that this is somewhat time-consuming, and to some it is annoying. Those who are ignorant of the significance of the problems these children present often tell the mother that the child will outgrow it. The mother used to be told that the child would outgrow rheumatism, and now she is told the child will outgrow temper. When you tell a mother that, you are getting nowhere. Unless we do pay attention to these problems, no progress can be made. We must persevere no matter what the difficulty is. We are dealing also with a great many children who are not pathologic, but simply have a clumsy way of using their faculties. I have seen a whole series of cases of enuresis which has been entirely a reaction against something disagreeable in the child's emotional life. If we tell the child what is really the matter, it will not have enuresis. In one case it took six months to find out why the child had enuresis, but we finally reached the cause through the study of dreams, and then the bed-wetting stopped almost immediately after the matter was treated. What pediatrician would take the trouble to collect and study the dreams of a child for six months?

DR. CROTHERS: One thing seems to me perfectly evident, and that is that any possible form of psychiatry is time-consuming. Dr. Lucas spoke of the psychiatrist as a person who could spend two hours on a case. A psychiastrist is one who sometimes spends two years in studying a patient. Time is of small account to anyone interested in conduct. I know cases in which I have taken much more than two weeks to find what I was seeking. After you find the method that leads along the right path, you may spend a great deal of time following it up. This is better business than to see years of educational work broken down, and the parents know that months lost from school are expensive in the long run. I feel that if a child's orderly education is interfered with, there is a crisis, so let us find out about it. We find out by having people who have done adequate social work visit the school and talk with the family, the governess and maid; such an agent can often find what all the trouble is about. When I started out with this work, some people took it for granted that I was a psychiatrist. I resented this because I was no good as a psychiatrist. I have always said "tell the family I have no technic, but if they come in I will see what investigation can be made." After sending someone out, the talking with the teacher and with the parents, if it takes ten or fifteen hours, is time well spent. Sometimes, after we have studied the case, the only solution involves breaking down plans for going through a certain college or into a certain business. Parents cannot always see why the plan that worked in their own lives cannot be followed. You have to go through a long process. I have not the slightest sympathy with the physician who cannot take an interest in these cases because they cannot be fitted into an ordinary appointment book. Physicians of that type necessarily transfer these cases to others, who must establish a whole new series of loyalties because the first man is not willing to give time to the problem. The difficulty is obviously increased, for confidence already once given has to be reestablished. The original physician is throwing away assets which the psychiatrist may not be able to regain.

DR. KNOX: I think you will all agree that we have listened to a very helpful and illuminating discussion. We pediatricians have learned that we should know more of the underlying principles of psychiatry, so that we can more intelligently treat our runabout patients and their parents, and am sure that the psychiatrists realize too that they should perhaps be more familiar with

out pediatric problems. There is undoubtedly a large common field of action for these two groups.

It reminds me of the story of the two negroes who enlisted in one of our colored regiments during the war, and were sent to London. While there, they saw a Scottish Highland regiment on parade. Rastus turned to Moses and said: "What kind of people is these?" Moses replied, "Indeed I do not know; when I looks at the legs I think they's men and when I see their clothes they looks like women," whereupon Rastus replied: "I'll tell you what they is, they belong to that well known, fine Middlesex regiment."

It would seem to me that we need a large number of additional recruits from both groups in this Middlesex regiment.

FIFTH SESSION

A Simple Dressing for the Breasts of Mothers of the New-Born Infants. Presented by Dr. L. R. DeBuys.

In the service of the new-born at the Touro Infirmary in New Orleans, the supervision of the food supply of the infant is the responsibility of the pediatric department. The mother's breasts are therefore cared for by the pediatrician. In this way the evolution of the nursing breast has been studied, beginning from the time of birth. There has been no fissuring of nipples, no caking, nor abscessing of the breasts during the five years that the service has been operating under the present plan.

No attempt is made to describe the care of the breasts and nipples in this paper; our object is merely to discuss the dressing and to describe its application. After much experimentation, the present dressing of the breast was evolved. It consists in the application of adhesive plaster in such a way as to support the breast, without making any undue pressure, perpendicularly to the chest wall and directly in front of the mother. By applying the dressing properly there is no obstruction to the ducts, no twisting of the nipples, nor compression of the mammary glands. In this way the milk flow may take place without restriction. Relief has been experienced by the mother in every instance in which the dressing has been applied.

In the application of the dressing the patient is placed in the recumbent position, and after the arms are elevated above the head the adhesive strips are put on. (1) A strap is applied from one side to the other below the breasts; (2) another strap is applied above the breasts, also extending from one side to the other; (3) a third strip is applied extending from beneath the breast to above the other breast; (4) a fourth strap is applied in the opposite direction from the third one, and (5) a last strap is placed in the median line extending from below the first strap to above the second strap. The size of the straps may vary, but are usually from 2¾ inches to 3¼ inches. The thickness of the adhesive straps depends on the size and slope of the breasts to which they are applied. The dressing was described graphically by lantern slides showing the various steps in the application of the dressing.

DISCUSSION

Dr. Carr: It must be a relief to the obstetrician to have the pediatrician take over these cases. The type of dressing for the breast is subject to consideration as to whether the mother has pendulous or large breasts. We know that the breast is suspended from the aponeurosis that covers the pectoral muscles, and some breasts can be kept in good position by a dressing that

is a little less elaborate. A strip attached over the shoulder and down to the nipple with one binder of a cross belt form may be sufficient. This obviates the use of much plaster which is sometimes uncomfortable and keeps the skin tender. It does not interfere with the care of the nipples and the usual routine of the nursing period. Certainly it is a great help if we are allowed to suggest and supervise the care of the breasts when we are taking care of babies in the nursery.

Dr. DeBuys: Dr. Southworth has asked me a question. I will first respond to Dr. Carr's remarks. First, as to the width of the adhesive strips, this varies depending upon their location. The lower one is 3¼ inches wide, the upper one 3 inches and the cross strips are smaller than that; the upright strip is 2¾ inches. The type and size of the breast will influence the size of the adhesive plaster. Sometimes we use, one, two or three thicknesses. In another paper which I read in Kansas City where Dr. Neff invited me a few years ago, I said something about the types of breasts. I classified them according to shape, size and function, and I thought we got a great deal out of that. In this paper I made no attempt to go into the care of the nipples, as that is a subject that requires special consideration. I disagree with Dr. Carr about the binder. We first used the binder and I saw the mother suffer a great deal. The breasts are not held up and are drawn together, and the mother is further made uncomfortable by having the binder across her back. It is hotter, and in applying this binder the nipples are buckled over and the ducts cannot fill out when the milk is in excess.

Eczema in Infants and in Young Children. Presented by Dr. Charles Gilmore Kerley.

Eczema in infants is a disease that is treated largely by the pediatrician and the general practitioner, owing to the fact that it is a constitutional disorder, the skin lesions being the outward manifestation of the abnormal processes within. It may be cured by removing the offending food substance from the child's nourishment. It is my belief that it is the protein factor in the food that supplies the irritant, and that there are other food factors, particularly cow's milk, fat and sugar, that enhance the protein sensitization or prepare the way. The scratch skin or intradermal tests are of practically little value in showing the way because the child may be but remotely sensitized. When a markedly eczematous breast fed infant is removed from the breast and given food of another nature, the eczema may subside and this proves that the irritant agent has been removed. When an eczematous infant fed on cow's milk fails to react and is given a substitute such as goat's milk, a cereal carbohydrate and vegetables, the eczema may subside and something has been proved. When an infant with fulminating eczema fed on raw cow's milk is given the same formula but boiled for six hours and the eczema subsides, it means that the irritant agent has been killed by the prolonged boiling. The remarkable change that has occurred in cases of eczema after the milk was boiled with cereal for six hours can be attributed to nothing else than the prolonged boiling of the milk, as the local treatment was negligible. In spite of the unreliability of the skin test in showing protein susceptibility in eczema, I employ it in every patient coming under my care, and find a reaction to one or more of the proteins in about 50 per cent of the cases, the order of response being first, egg albumin, second, fresh cow's milk and third, wheat protein. In the breast fed an improvement in the intensity of the eczema usually follows on making the mother a vegetarian, but the infants are rarely

cured. For the mother, all animal food is discontinued, including flesh, fish and fowl, and a saline laxative is given sufficient to produce two evacuations daily. A laxative sufficient to produce the same effect is also given the child.

If the condition is one of malnutrition, a test diet is given, consisting of evaporated milk, rice and a small amount of cane sugar. Rice is used because the child is less likely to be remotely sensitized to its protein. For children of the runabout age who can take other foods, a milk substitute made from dried fat-free milk with the addition of starch and saccharine has been used with marked success. Eczema in the runabout child in the great majority of cases is of animal protein food origin. The prolonged cooking of the milk is also carried out in these cases. Local measures alone, including the violet rays and roentgen-ray, are of little service. Four drugs that are of distinct value are salicylic acid, sulphur, tar and resorcin.

DISCUSSION

Dr. Koplik: I do not know of any subject that is more puzzling than the management of eczema in children. I am glad to hear about boiling skimmed milk. I do not believe all eczema can be classed together. In the Vienna Clinic, I believe it was thought that eczema was a local infection of some kind, which is true in some cases but not in all. Some time ago Finkelstein published an article claiming that fat caused eczema, and then some time later he repudiated that theory. Then there are other forms when a parasite underlies the eczema. I had that illustrated in a child who had been under treatment for months. The eczema was treated locally and with a good diet, and the eruption cleared up wonderfully. I do not use the skin tests in infants. I do not want any anaphylaxis in my office. In treating eczema, we are very much in the dark. I had a case the other day in a child of an exceedingly intelligent family, whose physician had put the patient on a skimmed milk diet. I ordered a different diet, and the condition improved. If eczema is due to protein, why does the condition localize on the face? With some forms of eczema children have asthma, and particularly when it is located on the arms and legs at the joints. In other words, there are different forms of eczema.

Dr. Blanco: I am interested in eczema because it is very common in the Argentine, and nearly all the infants are breast fed. I have tried to improve them with general treatment, but in the last two years I have had very good results with the following ointment: Bismuth subgallate, 20 Gm.; potassium sulphate and oxyquinolin sulphate, each 20 Gm.; hydrous wool fat, 800 Gm. and petrolatum 200 Gm. In one month, and usually in two or three weeks, the eczema clears up. It is difficult to treat eczema in the summer, for the children have other disturbances worse than eczema.

Dr. Talbot: I think eczema is another example of changing ideas in modern medicine. It was not so long ago that acidosis was considered a disease; now it is no longer considered a disease but a symptom, so with eczema; it is not a disease but a symptom, and a symptom which may be the manifestation from an external cause or from an internal cause. We must conclude that there are many possible reasons for eczema. It takes more imagination and more knowledge of the case to get successful results than in anything else I know of, barring asthma, which is the same thing in many ways.

Everyone remembers the interest of the dermatologists in the treatment of eczema with thyroid extract. I had a case of general eczema and found a high basal metabolism. The dermatologist insisted on giving thyroid. The child did not improve. In chatting with the mother, she said the child would not

take eggs, and that every time she took egg she vomited. We eliminated egg from the diet and the eczema cleared up. It is necessary to get all the details in the care of eczema. In another case with a marked dermatitis of the eczematous type, the child was found to have a lowered basal metabolism. In that case without other treatment the eczema cleared up on thyroid extract. Over dosing with thyroid was followed by a return of the skin symptoms, and on reducing the dose to a point that the basal metabolism was normal, the skin remained cured.

DR. HEIMAN: I believe we are all mindful of the fact that when we see a child with eczema, it has passed the first stage, the urticarial or stage of dermatitis. There are eczemas due to internal and also to local causes. I do not think it is necessary to give the children the sensitization tests. The majority will get well after 1 year of age. They are usually able to desensitize themselves. It is most important to stop the itching, and that cannot be done by salves alone. In our experience a preparation of coal tar, zinc paste N. F. IV and hydrous wool fat equal parts, has been found useful. Another point to remember is that after applying the salves and rubbing the face and body, a linen sheet, smeared with zinc salve, should be spread under the affected surfaces, and the hands and feet tied. Few mothers are willing to cooperate in this procedure, and unless they are willing to subject the child to this treatment we are unable to cure the eczema. Silk, wool and furs are irritating, and from the time a child is 5 months old it puts its hands in its mouth and rubs saliva over its face. In these cases tying the hands and feet and local applications have proved useful in our experience. I have never changed the food. If you give the children skimmed milk it is difficult to make them gain. As Dr. Blanco has said, most cases occur in breast fed babies; that has been our experience.

DR. O. M. SCHLOSS: There seems to be very little question that the forms of dermatitis called eczema can be of allergic origin. At the present time, however, there is not sufficient evidence to indicate that eczema is always of such origin. The relationship between eczema and allergy can be summarized very briefly.

As pointed out by Dr. Kerley, a large percentage of patients with asthma either suffer coincidently with eczema, or give a history of having had eczema at some previous time. It can also be demonstrated in certain instances that eczema may be directly due to some type of food, pollen or animal emanation. That eczema may be due to pollens can be proved not only by cutaneous reactions, but also by the seasonal incidence of the eruption and the therapeutic effect of removing the patient from the influence of the particular pollen to which he is sensitive, or by inoculation. There are some cases of eczema in which food is definitely implicated. For example, I have seen cases of eczema due definitely to egg, with prompt disappearance of the eruption when egg was removed from the diet. I have also observed similar instances in which milk was the offending food. Cases in which there is a clear cut relationship between food and eczema are unfortunately few in number, but the occurrence of such cases serves to demonstrate fairly conclusively that eczema may be an allergic disturbance, although they do not necessarily prove that it always is.

As far as the value of cutaneous tests in the investigation of eczema is concerned, they are just of sufficient value to warrant their use in obscure cases of eczema. In most instances they are useless, due either to the fact that no reaction occurs or to the fact that multiple reactions are elicited. One encounters cases occasionally, however, in which cutaneous tests give a valuable lead.

The problem of eczema in the breast fed infant is interesting. A number of investigators have believed that in the mother's milk may be secreted antigenic proteins which not only sensitize the infant but also produce an eczema. O'Keefe and Shannon both believe that the elimination from the mother's diet of articles of food to which the infant reacted was followed by improvement in infantile eczema. Shannon also believed that he could demonstrate, by anaphylactic tests, this antigen in the milk of mothers. Similar experiments, however, by myself and by Stuart did not confirm the work of Shannon. Whether there is secreted in human milk antigenic protein in amounts too minute to demonstrate by anaphylactic tests but yet sufficient to sensitize and intoxicate an infant, is purely a speculative question. There is one other possibility in regard to eczema of breast fed infants; namely, that the casein of human milk has apparently little, if any, species specificity, so that it is possible that an infant might become sensitized to the casein in its mother's milk.

Dr. Hand: I wish to thank Dr. Kerley for his addition to our armamentarium in treating eczema. Several years ago, I learned indirectly from a country physician that cow's milk may be the offending agent for some cases of eczema. An infant whom I had seen once in consultation, making all the suggestions I could, was taken away for the summer, and the local physician when called in stopped giving cow's milk; the eczema is said to have disappeared. But I must also agree with Dr. Koplik in looking on eczema as protean, both in its manifestations and in its etiology, and therefore I am thankful to Dr. Kerley for the expedient of the prolonged cooking of milk as another string to the many in our bows we need in managing this troublesome condition. As a result of personal experience, the first food change I make in the diet is to eliminate orange, grapefruit and oatmeal, with apparent benefit in some cases.

Dr. Charles Hendee Smith: Acute infantile eczema is so often complicated by extreme irritability of the skin that we obtain pseudoreactions to every scratch, and this may render the protein tests useless. The necessity of a careful history is very important, as suggested by a previous speaker. For example, I saw a child with eczema due to egg who would not get well, and I found that he was eating arrowroot crackers every day. On writing to the manufacturers of these crackers I found that there was 12 pounds of egg powder in every 100 pounds of the crackers. They were eliminated from the diet and the child showed a rapid improvement. Another factor which has not been mentioned is overfeeding. This may not cause eczema in all infants, but in one with a sensitive skin it may do so. Again a careful history often elicits the story that the child gained very rapidly at some time in the past. This period of rapid gain may be only a week or two, but it may start an attack of eczema which lasts several months.

In infants with an acute dermatitis it is difficult to keep local applications in contact with the skin, as the children rub them off and many mothers will not keep a mask on. Some children are irritated by grease in any form. I have used a mucilage with a chondrus base, which has the advantage of drying on the skin. Most preparations are slightly acid, but by mixing 2 or 3 drachms of magnesia magma the acidity is neutralized. Zinc oxide, from 2 to 3 drachms to the ounce may be added. It smarts for a short time while drying, but immediately after that it gives an airproof dressing which can be left on indefinitely. It is soothing, and works well in acute cases which do not need stimulation. A few drops of phenol (carbolic acid) can be added. After a

day or so the dressing may crack and come off in places, but more can be put on. It is also water-soluble, and can be easily removed. I have found that it helps in relieving the itching of the dermatitis, and that is a great deal.

DR. KERLEY: The object of my paper was to give a summary of my observations and conclusions regarding eczema in children, of whom I have had occasion to treat a vast number.

Briefly, eczema in infants is a manifestation of protein sensitization. This sensitization may be increased by the use of cow's milk fat and sugar.

The scratch skin or intradermal test is not to be depended on. A child may be remotely sensitized, sufficient for the production of a dermatitis, and yet fail to react to the skin test.

Proteins that produce the dermatitis may be rendered harmless or less harmful by the application of prolonged or extreme heat.

Local measures in management such as the roentgen ray, ultraviolet ray and local applications are of but temporary value. Patients apparently cured by these procedures means that the child has become immunized to the specific protein or proteins that caused the disease.

Patients are seen now and then in which the clothing worn is responsible for eczema; both silk and wool garments have caused the disease in infants and young children under my care.

TUBERCULIN SKIN REACTIONS. Presented by DR. CHARLES HENDEE SMITH.

In the Children's Medical Division of Bellevue Hospital, both Pirquet and Mantoux tests were used on all children for several years. The results are shown in table 1 (3,112 cases, 1921-1924). The same relative proportion obtained during each of the four years, about twice as many children reacting to the Mantoux as to the Pirquet. The Pirquet has therefore been discarded as an uncertain test, not worth the time it takes.

TABLE 1.—*Relative Value of Pirquet and Mantoux Tests*

Age	Total Cases	Pirquet Positive, per Cent	Mantoux Positive, per Cent
0 to 6 months	594	1.35	5.21
6 to 12 months	394	3.3	8.4
1 to 2 years	417	5.0	8.6
2 to 3 years	247	4.45	11.3
3 to 4 years	202	5.95	11.9
4 to 5 years	169	5.9	14.7
5 to 6 years	146	10.25	17.7
6 to 7 years	192	13.7	26.4
7 to 8 years	132	12.8	26.2
8 to 9 years	150	8.8	25.0
9 to 10 years	130	15.4	37.0
10 to 11 years	153	21.0	38.0
11 to 12 years	132	25.8	45.5
12 to 13 years	55	(18.2)	45.5
	3,112		

Total cases positive Pirquet, 242, or 7.8 per cent; total cases positive Mantoux, 517, or 16.5 per cent.

Mantoux used a solution of old tuberculin, 1:10,000, injecting 0.1 cubic centimeter or 1/100 mg. into the skin. This amount is rather small, and there is no objection to a larger dose, provided no general febrile reaction is set up. Most observers use 0.1 cc. of a 1:1,000 solution (0.1 mg.) which is a safe initial dose. On account of the severity of the local reaction in some cases, (especially extensive lymphadenitis, bone disease, etc.) it is better to use a smaller initial dose.

After trying various plans we have adopted the schedule shown in table 2.

All children are retested once if negative to the first test. Only suspected cases of tuberculosis are given larger doses if negative to the second test. Positive reactions to later tests are exceedingly rare, and occur mainly when the sensitivity is lowered by overwhelming tuberculosis. Fever may suppress the reaction temporarily (measles, etc.). Inert solutions, careless injections or reading are the only other sources of error.

The main objections to the Mantoux are the difficulty of making the dilutions and the rare severe local reactions (vesiculation or even sloughing). The dilutions may readily be made, however (namely, boil 100 cc. water, add salt

TABLE 2.—*Schedule of Tests with Old Tuberculin Solution*

Test 1:	0.5 cc.	1:1000 solution	=	0.05 mg.
Test 2:	0.2 cc.	1:1000 solution	=	0.2 mg.
Test 3:	0.1 cc.	1: 200 solution	=	0.5 mg.
Test 4:	0.2 cc.	1: 200 solution	=	1.0 mg.
Test 5:	0.2 cc.	1: 100 solution	=	2.0 mg.

TABLE 3.—*Percentage of Positive Reactions (Mantoux)*

Children's Medical Division, Bellevue Hospital (Ward Cases)

January 1921—July 1925

Age	Total Cases	Percentage of Positive Reactions	
0 to 6 months	673	4.75	
6 to 12 months	487	9.45	
1 to 2 years	503	9.35	
2 to 3 years	328	15.5	
3 to 4 years	245	13.0	14.5
4 to 5 years	210	16.2	
5 to 6 years	193	17.0	16.2
6 to 7 years	219	27.8	
7 to 8 years	167	32.9	30.0
8 to 9 years	222	27.0	
9 to 10 years	192	31.8	29.2
10 to 11 years	169	40.7	
11 to 12 years	169	43.7	42.3
12 to 13 years	55	45.5	
	3,832		

tablet and 0.1 cc. old tuberculin). The New York Health Department makes an outfit exactly similar to that for the Schick test, which makes the test readily available in hospital, office or at the bedside.

The test is not quantitative, and seems to depend on the size of the wheal of the injection as much as on the strength of solution or amount of tuberculin injected. Thus 0.1 cc. of 1:10,000 (0.01 mg.) gives nearly as great a reaction in a sensitive child as 0.1 cc. of 1:1,000 (0.1 mg.), while 0.01 cc. of 1:1,000 (0.01 mg.) gives a much smaller reaction than either. A control is not necessary, since no other substances give a similar reaction. About 1,000 cases were tested with broth concentrate prepared exactly like tuberculin, but none of these controls reacted. Tuberculin is not thermolabile and resists boiling indefinitely, therefore syringes and needles should not be used for Schick or other tests after being used for tuberculin.

Table 3 shows the total number of reactions obtained on all children who remained in the hospital long enough to have tests read. It probably represents

the amount of infection in the population of the East side of New York. The percentage of positive reactions is much lower than early European figures, but corresponds closely to later series tested by the same method in Germany, Denmark and Sweden; as well as to a series from schools of Ontario. Many other American series are not accurate because not all children were tested, but merely tuberculous suspects or contacts, which give a high incidence of infection.

When the Pirquet test is used, an error in the other direction results. Therefore the series in which both these factors of error occur are worthless. For statistical purposes it is impossible to judge to what extent these errors balance. There is a general misconception in the minds of physicians as to the percentage of positive tuberculin reactions, probably based on the early European figures. Pediatricians should stress the importance of the test, since the children who are infected need careful watching through the growing years, and should be helped to withstand the stress of early adult life when active tuberculosis commonly comes on.

DISCUSSION

DR. BLANCO: In Buenos Aires, a city of 2,000,000 inhabitants, we have made the Mantoux tests in about 4,000 children between the ages of 6 and 13 years. While the Pirquet test gave only 44 per cent of positive reactions, the Mantoux test gave 77 per cent. Negative cases were tested three times before they were recorded as negative. After this experience we have discarded the Pirquet test, and do only the Mantoux test as it is more sensitive.

DR. SISSON: Dr. Smith's discussion is very interesting. We have made the Mantoux test and our observations are very similar to those of Dr. Smith. One of the great aims in this work is the attempt to show the incidence of tuberculosis in childhood and early infancy, and then to follow the children through a number of years. My suggestion is that Dr. Smith repeat these tests after five or six years and compare his results with those he has just reported. As Kraus says, it is impossible to obtain a positive test in a subject who has not the disease. If we get a positive test with immunization, it is impossible to have tuberculosis later on. One or two points occured to me during the discussion. I did not understand how much emphasis was placed on a repetition of the test. We have felt that it is very important to repeat the test, using up to 1 mg. of tuberculin. It is definitely established that there is no reaction whatever in 1:1,000 and 1:10,000 dilution. A plus reaction means actual infection. It is our custom to repeat the test up to 1 mg. tuberculin, because many react negatively to higher dilutions and show a positive test with lower dilutions. We have the Mantoux test carried out in private practice, and get only about 50 per cent positive reactions, less than in hospital practice. Perhaps these children are in a more dangerous condition than those coming to the clinic. In my son, up to the age of 9 years there was no reaction, and I have thought that he is in a much more precarious condition than if he gave a plus reaction.

DR. SCHICK: I just want to say a word about the Pirquet and the Mantoux test, as we use to apply it in the Pirquet clinic and in the children's department of the Mount Sinai Hospital. It is generally admitted that a great deal depends on the technic that is used in giving the Pirquet test. This test is frequently made by those who have not had enough experience in giving it, and as they do too little scarification, they get no reaction. We know that if we make an original Mantoux test with a solution of 1:10,000 on one arm, and a Pirquet test on the other arm, the results of both tests are always the same,

the reactions being either both positive or negative. This proves that in spite of using concentrated old tuberculin in the Pirquet test, only about 0.00,001 tuberculin is absorbed. We have established the principle to first make the Pirquet test, and then if the Pirquet test is negative, after the elapse of twenty-four or forty-eight hours, we make the Mantoux test, using 1:100 solution, which means 1 mg. old tuberculin. This procedure gives us very satisfactory results. The preparation of the solution 1:100 can be made in a very simple way. We take 5 cc. physiologic sodium chloride solution; 1 cc. being 20 drops, 5 cc. is therefore 100 drops. We then add 1 drop of concentrated old tuberculin. From this solution 0.1 per cent is injected. The great difference in the results of the Pirquet and Mantoux test obtained by Dr. Smith may be due to the fact that he used tuberculin of lower strength. We had the same difficulties after the war on account of the unsatisfactory manner in which old tuberculin was manufactured. Even a clinically clear case of tuberculosis (spina ventosa, etc.) gave a negative Pirquet reaction. With the use of stronger tuberculin, the results improved. If Dr. Smith would concentrate his tuberculin by boiling, he would get smaller differences between the Pirquet and Mantoux test.

Dr. Helmholz: Just as Dr. Schick has indicated, we use the Pirquet test and follow it with 0.02 mg. intracutaneously, 0.1 mg. and finally 1 mg. if tests are negative. We have had the opportunity to see cases of tuberculosis that showed that there was an increased tendency to reaction by injecting larger amounts of tuberculin. We observed three cases, one of Pott's disease with complete healing and ankylosis, one of complete destruction of the entire head and neck of the femur, and one case of miliary tuberculosis with calcified nodules throughout the entire lung without any past history of illness. In all of the three cases the Pirquet test was negative, the intracutaneous test with 0.02 and 0.2 mg. was negative, but the test with 1 mg. was positive. In contradistinction to Dr. Smith, I think that with increased dosage a negative test may become positive, and when this occurs the Pirquet test also will be positive if repeated.

Dr. Freeman: In hospital work, in which the tests are made hurriedly, we often get negative results which I have thought might be due to the fact that the tuberculin has been rubbed off. Therefore we resorted to the Mantoux method, but this had the disadvantage that there was no control with which it could be compared. We did not obtain more positive reactions with the Mantoux test than with the Pirquet. We resumed the Pirquet test on account of the control with which it could be compared.

Dr. Hamill: In 1907, we had the tuberculin test carried out in a series of children in an institution for foundlings and orphans to determine the incidence of tuberculosis. As there were a number of active cases, we found, as we had anticipated, a high percentage of reactors, amounting to 78.5 per cent. This figure included some of the definitely tuberculous. As the Pirquet and Moro tests had but recently come into use, we made a comparison of these with the Calmette test in 132 of the cases. The technic was very carefully supervised with each of the tests. The results were almost exactly uniform. Since that time I have used the scarification test with entire satisfaction. I am convinced, however, that a proper technic is essential to success. In my hospital wards I find that my internes get more uniform results with the intracutaneous test until they have developed a good technic in the Pirquet, when their results with the two are uniform.

Dr. Charles Hendee Smith: I am not sure that the evidence is complete that it is desirable to have positive reactions in children. I feel much more

comfortable in regard to my child because he has not a positive tuberculin reaction. The early infection does not immunize the child who develops tuberculous meningitis or a bone lesion. There are reasons to believe that the longer we can postpone a positive reaction the better. Most students of tuberculosis agree that the "army of the adult tuberculous is recruited from those infected in childhood." Therefore, the early recognition of the presence of infection as shown by the positive skin test enables us to try to forestall the development of serious active disease.

In this series, all children who failed to react to the first test were retested one or more times if they were in the hospital long enough, giving up to 1 mg. of tuberculin in suspected cases. There were very few who reacted to the retests — so few that we feel that the first test is quite reliable. Occasionally when the chart shows a negative first test and on the second test a positive reaction, we go back to the original area and find pigmentation, showing that there was probably a faulty reading. The intern makes a drawing of the size of the reaction, so that there is less chance of error in recording "positive or negative."

I think Dr. Schick is right in what he said about the technic of Pirquet. This is the whole story. For example, recently I made twelve Pirquet tests on one child who had only a slightly positive Mantoux reaction, and all were positive. However, Pirquet himself obtained twice as many positive reactions on retesting, showing the uncertainty of the method. We are looking for a practical test on which we can rely when done as a routine by the average hospital staff, and when the Pirquet test does not work. There is too large a factor of error.

I think Dr. Schick's plan of performing a Pirquet and then a Mantoux test with 1 mg. of tuberculin is dangerous to turn over to a hospital service. With the factor of error inherent in the Pirquet, which depends on the method itself rather than on the sensitiveness of the child, it seems to me too great a risk to give an initial dose of 1 mg. instantaneously. I have even seen general reactions following the Pirquet test, with a temperature of 104 F., and a good sized slough. In private practice, the plan of making a preliminary Pirquet test has a distinct disadvantage. One has to obtain the parents' consent to make a tuberculin reaction. If a Pirquet is made and is negative, then the physician has to tell the parents that the test is not conclusive and prolong their anxiety while waiting for the result of a second test. He is in an awkward position in trying to explain to the laity the reasons for using an inaccurate test when a more accurate one is available. In hospital work the preliminary Pirquet test wastes time, since many children do not remain long enough to be retested accurately with the Mantoux test.

CERTAIN EFFECTS OF AMMONIUM CHLORIDE INGESTION ON CALCIUM METABOLISM. Presented by DRS. S. G. ROSS and JESSIE BOYD SCRIVER (by invitation).

The relationship of tetany to changes in the acid-base equilibrium of the body has been the subject of much investigation. In certain types of tetany, an alkalosis is present. In infantile tetany, the consensus of opinion is that there is no alkalosis. The characteristic blood finding in this type is a lowered calcium concentration in the serum. Calcium chloride, or ammonium chloride, both of which cause an acidosis after ingestion, have been found effectual in relieving the symptoms of tetany. Some investigators have found that ammonium chloride raises the level of serum calcium in tetany; others have found the reverse. The present investigation is an attempt to elucidate the action of ammonium chloride in calcium metabolism.

In a series of five cases of infantile tetany, the administration of ammonium chloride produced the following changes in the blood serum: (*a*) an increase in the calcium varying from 0.4 to 3.5 mg. per hundred cubic centimeters; (*b*) a reduction in the combined carbon dioxide; (*c*) a reduction in the inorganic phosphorus; (*d*) an increase in the chlorine; (*e*) no change in the total base. The extent of the increase in the calcium seemed to have a relationship to the extent of the reduction in the combined carbon dioxide.

The effect of diets containing large and small amounts of calcium was studied. On the low calcium diets, the increase in serum calcium, after ammonium chloride administration, was generally less than when a high calcium diet was given. When ammonium chloride was given, the output of calcium in the urine was less with a low than with a high calcium intake.

The effect of ammonium chloride ingestion on the calcium balance of the body was studied. Ammonium chloride always caused an increased excretion of calcium by the kidney. The output of calcium in the stools gave somewhat conflicting results. In most cases, the output was greater when ammonium chloride was given. This may be due to one of two factors: (*a*) increased excretion of calcium by the bowel wall, or (*b*) diminished absorption as a result of increased catharsis. Further work is necessary to decide this question. In either event, the experiments show that the salt increases the total output of calcium from the body. This finding, in conjunction with an increase in the concentration of calcium in the serum, makes it justifiable to assume that ammonium chloride acts by mobilizing calcium from the body reserves, rather than by increasing the absorption of calcium from the bowel.

The conclusions drawn from the investigation are that ammonium chloride mobilizes calcium from the body reserves probably by virtue of its acid-producing effect. The treatment of tetany by this salt is therefore an "acid therapy."

DISCUSSION

DR. KRAMER: Studies on the calcium and phosphorus concentration of the serum of rachitic children and animals have shown that the calcium may be low, the inorganic phosphorus concentration of the serum may be reduced, or the concentration of both of these elements may be below normal. Whenever the calcium is reduced, the normal level may be reestablished by the administration of ammonium or calcium chloride.

It was thought that in cases in which the inorganic phosphorus is normal and the calcium reduced, the restoration of the calcium to its normal concentration by the administration of calcium or ammonium chloride would restore the calcium phosphorus equilibrium, and hence healing of the rachitic process should follow. This failed to occur. Dr. Ross' work supplies an explanation for the failure, since it shows very clearly that ammonium chloride raises the calcium concentration in the serum by mobilizing calcium from the tissues and presumably from the bones. This treatment should aggravate rather than ameliorate the rachitic process. It also explains why the administration of ammonium chloride must be accompanied by cod liver oil or by some other antirachitic agent, if the results are to be permanent. For with the discontinuance of the ammonium chloride therapy, the calcium of the serum again drops below normal and the symptoms of the tetany reappear.

Book Reviews

STANDARDS FOR THE DIAGNOSIS, CLASSIFICATION AND DISPOSITION OF CASES OF PULMONARY, AND GLANDULAR TUBERCULOSIS. Ed. 6. New York: National Tuberculosis Association, April, 1926.

In this booklet are briefly analyzed the essential criteria of diagnosis of pulmonary, hilum and gland tuberculosis, together with the minimum standards on which such a diagnosis should rest. A scheme is also set forth for a uniform classification of the various stages of the disease.

A surprising amount of information concerning symptomatology, physical diagnosis and the interpretation of roentgen-ray films and tuberculin tests is to be found in this little pamphlet of 31 pages. Copies may be had free of charge by any physician on request to his state tuberculosis or public health association.

TECHNICAL GUIDEBOOK FOR THE CARE OF CHILDREN. By DR. B. DE RUDDER. Berlin: Julius Springer, 1926, pp. 68.

This little handbook for nurses is full of useful information regarding the immediate care and nursing of sick infants and children. Every detail is covered, and the directions are explicit. There are special chapters on the clothing of infants and children; on breast feeding and expression of breast milk; and there are detailed directions for the preparation of most of the artificial foods. The various baths and packs are well described, and directions are given for the preparation of all special treatments such as intravenous injections, surgical procedures, tracheotomies, etc. One of the first general rules is quite apt: "It goes without saying that every direction of the physician shall be carried out to the last detail. Inquiry should be made regarding any order not clearly understood."

LES FEUILLETS DU PÉDIATRE. By G. BLECHMANN, formerly Chief of Clinic at the Hospital for Sick Children, Paris. Ed. 1. 8 Place de l'Odéon, Paris: G. Doin and Co., 1926.

This small paper-bound volume of 150 pages does not warrant a long review. In the preface, which is written by Dr. Noir, it is stated that the pediatrician must be an artist as well as a student, and he must be able to utilize knowledge most efficiently. The book then proceeds in short chapters, which deal largely with drug therapy of various disorders such as tetany, enterocolitis, convulsions, impetigo, pertussis and the like.

In miscellaneous chapters scattered throughout the book in an aimless manner, are notes on bathing, condensed milk, preparation of vegetable soups, mercurial inunctions, the use of calomel and a number of other subjects. The reviewer feels that this book has little value, certainly to the American pediatrician.

CLINICAL PEDIATRICS, DISTURBANCES OF NUTRITION AND GROWTH. By P. NOBÉCOURT. Price, 36 francs. Pp. 404, with 104 illustrations. Paris: Masson et Cie, 1926.

This is the third volume of Clinical Pediatrics by this author, the two preceding ones being on Respiratory Disease and Circulatory Disease. The present volume treats especially the clinical features resulting from endocrine

disturbances, almost half the text being taken up with a discussion of thyroid disturbances and diabetes. In addition, are treated the various statural hypertrophies, obesity, "hypotrophies," in which are included hypoplasia and atrophy, with a chapter on chronic arthritis and fragile bones in infancy.

The plan of considering each subject as, for example, myxedema, is to give first, a most complete and detailed report of the clinical examination of one or more typical cases with photographs, roentgenograms, charts, anthropometric measurements, blood studies, etc. Then the various deviations from the normal in these cases are discussed and correlated with a final general résumé of the condition and the therapy.

The volume offers an unusually complete collection of clinical data on the subject considered, and should be of great service to those who are interested in such detailed observations. The style of presentation is good, and even though one may not always agree with the author's conclusions, his point of view is of interest. In the discussion of the treatment of diabetes, the considerable space devoted to the handling of cases without the use of insulin, is more of historical than of practical value.